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A TEXT-BOOK

OF

DISEASES OF THE CHEST

PERICARDIUM, HEART, AORTA, BRONCHI, LUNGS,
MEDIASTINUM AND PLEURA

BY

EGBERT GUERNSEY RANKIN, A.M., M.D.,
PROFESSOR OF THEORY AND PRACTICE OF MEDICINE, NEW YORK HOMŒOPATHIC MEDICAL COLLEGE, PHYSICIAN TO THE METROPOLITAN HOSPITAL, DEPARTMENT OF PUBLIC CHARITIES AND TO THE FLOWER HOSPITAL, NEW YORK

WITH 63 ILLUSTRATIONS

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1905

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1905

THIS BOOK IS INSCRIBED

TO

A. E. R.

PREFACE.

THE effort of the author is to present a work which is in full accord with the latest advances in modern pathology, bacteriology and all channels of medical research.

Notwithstanding the advantages which a long hospital service naturally affords for the observation of disease, such forms but a part of the basis of this volume, the works of many having been diligently studied and compared.

The discussion of treatment is on broad lines, embracing adjuvant measures and those remedies which experience has proved valuable, the endeavor being to present the same in a thoroughly practical manner.

A feature of the book is the recognition of the X-ray as an important adjunct in the diagnosis of thoracic diseases. For the valued section on this subject by Dr. William H. Dieffenbach warm thanks are expressed.

The author also desires to thank Mr. Charles Brush, Electrician of the Metropolitan Hospital, for the photographs and skiagraphs for the half-tones from actual specimens and subjects, and to call attention to the fact that with the exception of the sketches and one other illustration all were collected at the Metropolitan and the Flower Hospitals.

Indebtedness is acknowledged to Mr. J. Charles Arter for the original sketches made especially for the writer, to Dr. Guy B. Stearns and Dr. Ernest Thum for the sphygmographic tracings, to Dr. Edward Harris, Dr. Parry D. Saylor, Dr. John M. Hanna, Dr. Eugene F. Mills and Dr. E. W. Kellogg for clinical and pathological data.

E. G. R.

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ERRATA.

Pages 27, 6th line; 95, 22d line; 466, 3d line; 524, 14th line; 578, 30th line, read "agaricine" for "agaracine."

Page 30, 3d line, read "arsenis" for "arsentis."

Page 224, 28th line, read "dilatation with compensative hypertrophy" for "hypertrophy with compensative dilatation."

Page 314, 31st line, read "are" for "is."

Page 471, 18th line, read "pseudo-lobar" for "pseudo-lobular."

PART I.

DISEASES OF THE HEART.

SECTION I.

THERAPEUTICS OF CARDIAC DISEASES.

THE intimate and vital relation to other organs and to every portion of the organism which the circulatory system sustains necessarily entails in the treatment of affections of the heart the consideration of functional and structural disorders of all parts of the body. When once the blood-current, as it flows through the channels of the circulation, on account of some obstacle or deflection in its course, ceases to be evenly maintained, the blood tends to accumulate in the veins and to diminish in quantity in the arteries, and in consequence there ensues venous stasis with disturbance of all the great functions and ultimate structural changes in distant organs. In seeking, therefore, indications for treatment, not only the diseased heart but also the changes which it effects in other parts of the system must be taken into account.

Rest.—While as in other acute inflammations the importance of rest is well recognized and naturally enjoined, in acute inflammation of the heart it possesses peculiar claims which cannot be too strongly emphasized. Complete and absolute repose in the recumbent position, with the prohibition of all movement and exertion of any kind, by lessening the energy of the heart, secures to the inflamed organ a relative degree of rest and thereby tends to diminish the extension of the inflammatory process. Again, after the subsidence of the acute stages, when resolution has begun, rest is also of paramount importance. Here it plays the part of a prophylactic of probable sequelæ.

In acute pericarditis the weakening of the myocardial structures, which is especially marked if the inflammatory process has been excessive, demands that the greatest caution should be exercised lest the weakened walls be overtaxed and irreparable dilatation result. Therefore, rest should be enjoined until the general health and tone of the system have been well established. In the convalescence of acute endocarditis the question of rest obtains a twofold force, for here not only must the possibility of dilatation be guarded against, but also the danger of further damage to the valves. Under the influence of prolonged rest in bed some recently developed murmurs will disappear, while others will become less pronounced. In all instances of damage to the valves, rest is the most important factor in allowing the heart to adapt itself to the changed conditions and in favoring the development of compensation.

In chronic valvular disease rest is to be viewed in a far different light, as the patient does not as a rule come under observation until the equilibrium of the circulation has been disturbed, when exercise should be restricted and kept under careful supervision, rest being enjoined according to the demands of each individual case. It should, in all instances, constitute one of the most important factors of treatment. In advanced conditions the exigencies of the situation render its discussion superfluous.

Diet.—In acute lesions, light, easily digested nutritious food should be given in small quantities, at frequent intervals, as in other acute diseases. In chronic cases, generally speaking, the food should be small in bulk as well as highly nutritious, but more decided specifications are usually necessary. In cases dependent upon gout or a gouty diathesis, or associated with lithæmic states of the system, the regimen usually prescribed for these conditions should be followed. In those associated with renal disease the diet should be according to the rules required for interstitial and parenchymatous nephritis, according to the nature of the case. In conditions tending to obesity or depending to a large extent upon that condition, reduction of the excessive amount of adipose tissue should be attempted by diet and exercise, as suggested by Oertel.

The amount of fluids to be allowed in the dietary of chronic heart disease will be governed entirely by the nature of each individual case. When there are tendencies to venous stasis a restricted and dry diet should be prescribed. The use of alcoholic stimulants should be entirely withheld or limited. Except in elderly persons, who have been accustomed to their use all their lives, they are seldom necessary. When deemed desirable they are best taken with the meals. Abstinence from coffee and tea is often necessary, especially in the case of those who have indulged to excess in these beverages.

Abnormally high arterial tension, which in all instances must be combated, is in no small degree influenced by diet, especially in the instances of gouty diathesis and in renal disease.

In chronic renal disease, gouty conditions and uric acid excess, fluids, especially in the form of alkaline mineral waters, should be taken in considerable quantities. In every instance, whatever be the diet, all articles of food which tend in any way to give rise to digestive disturbances should be withdrawn. Indigestion not only interferes with both the general nutrition and that of the heart, but may cause mechanical disturbance by giving rise to gaseous distension of the stomach and bowels.

Excretory Functions.—Care should be exercised not to allow the bowels to become constipated. This is important, not only in order to avoid sluggishness of the portal circulation, but also to obviate the physical effort necessary to relieve the bowels when in this condition, which when compensation is feebly maintained is a matter of important consideration.

The functions of the kidneys should be watched for excess of solids, albumin and casts. The amount of urine must also be carefully watched, especially when large doses of digitalis are being administered.

The functions of the skin should not be neglected. Bathing daily should be the routine practice of everyone in health, not only for cleanliness, but as a maintenance of health. In diseases of the heart a sponging should be substituted for a tub bath, and in the case of those in weakened conditions this should be administered by an attendant, as the exertion otherwise may prove harmful. Persons suffering from cardiac affections should wear flannel next to the skin in winter.

Climate.—The tendency to bronchial inflammation and pulmonary congestion in those suffering from weakened hearts, and in those with valvular disease in whom compensation is maintained with more or less difficulty, renders the question of climate an important one. Sudden change of temperature and inclement weather should be avoided as far as possible. Whenever practicable, such persons should reside in a mild, equable climate, at an elevation of not more than two or three thousand feet. Some, on the other hand, do better at the seashore.

Sleep.—Insomnia is often a symptom of annoyance, and of serious consequences. Not only is the patient harrassed and distressed, but the general symptoms are aggravated and the process of nutrition suffers interference, thus adding materially to the progress of the malady. In conditions dependent upon reflex irritability, bromide of soda or potassium, ignatia, coffea, and sometimes passiflora incarnata will prove of service. When the insomnia is dependent upon cerebral exhaustion decided sedatives may be administered, such as trional, sulphonal and chloralamid. Frequently a glass of hot milk or a little easily digested food at bedtime will be helpful. Chloral is best avoided, as it is too depressing. Opium and its alkaloids in moderate doses are free from danger; their after-effects, however, are often undesirable, but they should not be resorted to until other measures have failed, except in acute conditions when they may be be occasionally administered with good results.

General State of the System.—Every effort should be used to maintain the general health, for the heart, like other organs, shares in a general decline. Anæmia shows a special disposition to the augmentation of valvular defects and may in itself give rise to relative insufficiency from dilatation of the walls of the left ventricle and thus seriously complicate existing lesions. Valvular disease in itself also tends to the deterioration of the blood. These facts should always be held in view in seeking indications for the treatment of cardiac diseases.

Exercise.—One of the most important questions which the physician has to decide is that of exercise. Whether or not it should be active or passive, and how far the patient should be allowed to go, recognizing in all instances the importance of exercise in tissue metamorphosis. Exercises best suited to the earlier stages of valvular disease are discussed in detail under that subject.

In more pronounced conditions for those who are able to go about, graduated active exercises are of service. The ascent of moderate inclines with frequent rests, in order to allow the patient to regain the breath, as recommended by Oertel, is suitable for some of these cases. In still more advanced conditions, especially when the patient is unable to walk much, gentle massage should be employed, but never sufficiently long to cause fatigue. The resistance exercise of Schott cautiously administered will also prove helpful.

The Oertel Treatment.—The object of this treatment is to stimulate the heart by exercise, carefully adapted to the condition and capacity of the patient, in order to induce hypertrophy and to diminish the volume of blood by restricting the amount of water in the diet and increasing that eliminated. The author of the treatment claims success in cases of cardiac dilatation, in valvular diseases even when compensation has been damaged, and dropsy and other symptoms of impeded circulation are present, and in fatty heart unattended with disease of the coronary arteries. The treatment consists of a series of graduated muscular exercises. The patient is required daily to walk a certain distance up a gentle incline; the pace and length of the walk are gradually increased each day. An altitude of two thousand feet should be selected for residence during the course of treatment. The diet is under careful supervision and the amount of fluids ingested is estimated.

The types of valvular disease where this method will prove beneficial are those of recent origin where compensation has been established and in those where it is maintained with difficulty. Its effects are more marked in cases of fatty heart without degeneration of the cardiac muscle (fatty infiltration), especially in obese patients addicted to excesses in eating and drinking. When this treatment is instituted great caution is necessary.

The walk up the gentle incline in the pure, partly rarified air is more conducive to the development of hypertrophy than on a level under less favorable conditions. In the case of the fatty heart this gentle exercise with the restriction of fluids tends to dissipate the excess of fat and tone up the heart muscle.

The Schott and Nauheim Bath Treatment.—This is the system of exercises and baths as instituted in Nauheim. The exercises consist of a series of simple movements of each limb, flexion, extension, abduction, adduction, and rotation, also rotation of the trunk. Each movement is made against a certain amount of resistance force systematically and with deliberation, with intervals of rest between each. Shortness of breath is a signal to discontinue. After resting they should be resumed.

The waters of Nauheim are distinguished by their richness in carbonic acid gas, by the presence of sodium chloride, calcium chloride and iron carbonate.

The following is a complete list of the exercises as given by Bezly Thorne, M.D.*

(1) Arms extended in front of body on a level with the shoulder-joints, palms of hands meeting in front of the chest. The operator places his hands on the outer surface of the patient's wrists in such a manner that the ulnar side of the patient's wrists rests in the fork of his own thumbs and forefingers. He places one foot in front of the other so he may lean forward, while the patient's arms are carried outward till they are on a line with each other and with the transverse diameter of the chest. The operator then places his hand, with a similar disposition of the thumb and finger, on the palmar surface of the patient's wrist and then offers resistance, while the hands and arms are being brought back to the first-mentioned position.

(2) The arm and hand of one side at a time are extended in the dependent position, with the palm directed forward, the operator standing at the patient's side places his open hand on the palmar surface of the patient's wrist, the thumb being only on the dorsal surface. The patient then flexes the forearm without moving the upper arm until the fingers come in contact with the shoulder. The operator then places the palmar surface of his own hand on the dorsal surface of the wrist and keeps it there, while the flexed arm is being extended to the position from which the movement commenced.

(3) The arms are extended vertically in the dependent position, palms forward; they are then raised outward until the thumbs meet over the head; they are now brought back to the original position. The operator faces the patient and resists the upward movement on the radial side of the wrist and the downward movement on the ulnar side.

(4) The hands, with fingers flexed from the end of the first phalanx in such a manner that the second phalanges of the respective fingers of the two hands are in opposition with their fellows of the

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* The Schott Method of Treatment of Chronic Diseases of the Heart. W. Bezley Thorne, M.D., 3d edition, London, 1899.

opposite side, are pressed together in front of the lower part of the abdomen. The thumbs are extended and lie within the three sides of a rectangle formed by the flexed fingers and touch each other at their tips. The arms are then raised until the hands are on a level with the top of the head. Resistance is made by placing the hand on the radial surface of the wrists. The movement is then reversed. Before the return movement is made the operator changes the position of his hands so as to receive the wrists in the fork of thumb and forefinger, the palmar surface of his fingers being applied to the palmar surface of the patient's wrist.

(5) The extended arms are placed in the depending position with the palms of the hands resting against the thighs. They are then raised in parallel planes until vertically extended. The movement is then reversed. The operator faces the patient, and in order that he may maintain a uniform and effectual resistance, the relation of his hands to the patient's wrists must pass through the following changes: In the first position the fork of the hand must be applied to the radial part of the wrist. As the arms rise to an angle of 45° to the body his fingers glide around the wrist until they are lightly folded around the radial surface of the wrists. Before the reverse movement commences he receives the ulnar aspect of the wrist in the fork of the hand. While the arms are descending his thumb moves outward, and at the same time the fingers glide around the dorsal surface of the wrist in a direction opposite to that which the thumb is taking, in such a manner and at such a rate that when the patient's arms are on a level with the shoulder the ulnar aspect of the wrist rests on a reversed fork formed by the radial aspect of the operator's forefingers and the thumb pushed out to a right-angle with somewhat flexed fingers. As the hand descends toward the thigh the tips of the operator's fingers gradually glide around to the ulnar aspect of the wrist, so as to resist the downward and backward movement of the arms. This is difficult for the operator.

(6) The trunk is flexed forward without the knees being bent and then brought back to the erect position. The operator stands at the patient's side with one hand on the upper third of the patient's sternum, the other hand supporting the mid-lumbar region. The reverse movement is resisted by placing one hand over the junction of the cervical and dorsal portions of the spine.

(7) The trunk is rotated without movement of the feet, as far as it can be carried to one side, to the right, then to the left, and lastly brought back, face forward, as at starting. The movements are resisted by one hand being placed in front of and a little above the advancing axilla, while the other is placed on the receding shoulder. The operator must, to a certain extent, move around the patient when the second stage of rotation is being performed, and will be able to do so most evenly and securely by carrying one foot around behind the other.

(8) The trunk is flexed laterally first to one side and then the other, and thirdly brought back to the erect position. The operator stands in front of the patient. When the movement is to the right, his left hand is pressed against the right side of the chest in the axilla, while the right firmly supports the opposite hip, and *vice versa.*

(9) This movement is the same as No. (2), with the exception that while it is being performed the fists are firmly clenched.

(10) The arms are flexed in succession, as in No. (2), with the difference that the palmar surfaces are turned outward and the fist is firmly clenched.

(11) The arm is extended in the depending position, the palm of the hand against the thigh, then a complete revolution is made from the shoulder-joint, forward and upward, until it is raised vertically alongside the ear. The operator stands at the patient's side with his fingers folded around the radial side of the wrist. His hand must be ready to receive the wrist whenever it reaches the vertical position, and to maintain the resistance until the arm has descended to the position whence it started. The movement is performed with one arm at a time.

(12) The arms are extended vertically in the depending position, the palms of the hands resting against the thighs. They are then moved upward and backward in parallel planes as far as it is possible, without bending the trunk forward. The upward movement is resisted with the forks of the hands on the ulnar aspect of the wrist; the downward, by folding the fingers around the radial surface.

(13) The patient stands with one hand resting on a chair or table, with the thigh of the opposite side flexed on the trunk to the extreme limit, and then extends it until the feet are side by side. The

leg should hang downward from the knee-joint. The upward movement is resisted by a hand placed immediately above the knee. The return may be resisted by a hand placed on the lower side of the thigh or under the sole of the foot.

(14) The patient supporting himself with one hand, as in the last movement, bends the whole extended lower extremity in succession, first forward to the extreme limit then backward the same way, and finally brings one foot alongside the other. The forward movement is resisted in front of and above the ankle, the backward movement behind.

(15) The patient supported in front by a chair or table stands on either foot in succession, the leg of the opposite side being flexed on the thigh. The upward movement is resisted by pressure on the heel, the return movement above the instep.

(16) The patient resting one hand on a chair and standing on the foot of the same side raises the extended lower extremities in succession upward from the hip-joint, and reverses the movement. The operator resists by means of one hand placed above the ankle.

(17) The arms extending horizontally outward are rotated from the shoulder-joint to the extreme limit, forward and backward. The movements may be resisted by the operator grasping the ulnar edge of the metacarpal portion of the hand, or by closing his thumb and forefinger in a ring around the wrist.

(18) The hands in succession are first extended then flexed on the forearm to the extreme limit, and lastly brought on a line with the arm. The operator's one hand supports the wrist, while the other resists the movement of the metacarpo-phalangeal junction, first on the dorsal, then on the palmar and then on the dorsal side again.

(19) The feet in succession are flexed and extended to the extreme limit, then are brought back to the natural position. The movements are resisted on the dorsal and plantar surfaces at about the level of the metatarso-phalangeal joint.

Each movement should be performed uniformly and evenly, none repeated twice in succession with the same limb. The patient should rest after every combined or single movement. No exercise or movement should be permitted to accelerate the respiration. The following are indications for the cessation of the movements: Dilatation of the alæ nasi, drawing up of the corners of the mouth, duskiness or pallor of the lips or cheeks, yawning and palpitation. If the patient is inclined to hold the breath during the exercises he should be directed to count. The limbs and whole body should be free from all constriction on the part of the clothing. It is not to be supposed that the patient is subjected to all these movements, or that any special order is essential. On the contrary, only a certain number are employed, and under certain conditions some must be omitted entirely, as in the instance when the patients are unable to leave the bed. Others, again, would prove harmful. These movements must, therefore, be applied with caution and after due examination of each case. They should in all instances be only applied by an expert and their effect should be carefully noted. Evidence of fatigue is a signal for their cessation.

The effect is in many instances marked. The rate of the pulse diminishes and its volume increases. The area of the heart dulness lessens, and in the case of dilatation the apex-beat returns toward its normal position.

Nauheim Baths.—The treatment of circulatory affections by means of baths is said to have been first advanced by Hope, of England, but it was not until comparatively recent years that the subject attracted the attention of the profession at large. Owing to the labors of Theodor and August Schott, Goedel and Beneke, the use of mineral baths, together with the system of exercises above described, has now attained an accepted position in the therapeutics of diseases of the heart, the system having been extensively elaborated at the springs of Nauheim.

Nauheim is a small German watering-place situated at the north-eastern extremity of the Taunus range, in the Grand Duchy of Hesse, at an elevation of four hundred and fifty-two feet above the sea-level. It possesses several springs, some of which are used internally and some externally; the latter are said to come from a depth of six hundred feet below the surface. Their temperatures range from 82° to 95° F. The waters contain, per 1000 parts, 20 to 30 parts of sodium chloride, 2 to 3 of calcium carbonate with smaller proportions of lithium, potassium, magnesium, strontium, barium, iron, manganese, zinc, bromine and arsenic. They contain, in addition, a large amount of carbonic acid gas, amounting in bulk to 1040 per 1000 parts, and in weight 4 : 1000. The waters, as a rule, are used at their natural temperatures.

The baths are employed as the simple saline, effervescent and effervescent current. The saline baths are administered in the strength of 15 parts of sodium chloride and 1 of calcium chloride to the thousand, at a temperature of 95° F., the carbonic acid gas being allowed to escape before use. The strength of the baths is increased daily and the temperature modified according to the demands of the individual case. The effervescent baths contain the full amount of gas and salts. The effervescent current baths are the same as the latter, with the exception that the water is allowed to run in and out of the tub during use.

The method of administering is to commence with the weak salt baths at a temperature of 95° F., which should not last more than five minutes. Afterwards, the baths are increased in strength, the temperature lowered and the duration lengthened. It is not usual to give the bath daily, but to let one or two days intervene. After a number of these saline baths have been given the second series or the effervescent baths are used. At first the temperature ranges from 92° to 95° F., and the duration from four to eight minutes. Gradually the duration is lengthened to ten to thirty minutes and the temperature lowered. Finally the effervescent current bath is employed.

During this course the waters of the Kurbrunnen, or the Karlbrunnen, are taken if there are any digestive disturbances. The entire course of the treatment continues for about six weeks. The effervescent baths are usually commenced after a fortnight, in some cases sooner; in those of a more severe type they must be deferred for a much longer period or not given at all.

For those who cannot leave home the artificial Nauheim baths may be employed with almost equally good results. The administration of these baths should be on the same general plan as that pursued at Nauheim. They also embrace a series extending over six weeks, including intermissions, beginning with about 1 per cent. of sodium chloride, 1 per cent. of calcium chloride and no carbonic acid gas. Following the same details of application pursued at Nauheim during the first week, the baths should be continued four or ten minutes at a temperature of 92° to 95° F., the proportion of solids gradually increased, the duration lengthened and the temperature lowered. About the second week of treatment ¼ per cent. of carbonic acid gas is added; this is also gradually increased to 1 per cent.

A scheme for a six weeks' course of baths would be about as follows:

First Week: ½ per cent. warm sea-salt bath (2 lbs. to 50 gals.). Temperature, 94° to 95° F. Duration, four to ten minutes. Intermission on third day.

Second Week: ¾ per cent. warm sea-salt bath (3 lbs. to 50 gals.). Temperature, 93° to 94° F.; four to ten minutes. Intermission on fourth day; ¼ per cent. carbonic acid gas.

Third Week: 1 per cent. warm rock-salt bath (4 lbs. to 50 gals.). Temperature, 92° to 93° F. Carbonic acid gas, ½ per cent.; eight to ten minutes. Intermission every fourth day.

Fourth Week: 1¼ per cent. bath salt, warm (5 lbs. to 50 gals.). Temperature, 91° to 92° F. Nauheim concentrated brine salts, 8 ozs.; ten minutes. Carbonic acid gas, ¾ per cent. Intermission fourth day.

Fifth Week: 1½ per cent. bath salts (7 lbs. to 50 gals.). Temperature, 90° to 91° F. Nauheim concentrated salts, 10 ozs.; twelve minutes or longer. Carbonic acid gas, ¾ per cent. Intermission fifth day.

Sixth Week: 2 per cent. bath salt (10 lbs. to 50 gals.). Temperature, 80° to 88° F. Nauheim concentrated salts, 12 ozs., and carbonic acid gas, 1 per cent.; fourteen minutes or longer. Intermission every fifth day.

The final step in the treatment is two or three weeks of rest at some place of moderate elevation. In successful cases the improvement is gradual and sometimes does not fully show itself until the patient has resumed his ordinary mode of life. The improvement may continue for many months and in some cases for more than a year.*

Immediately after the bath the patient usually experiences a sense of refreshment, followed by an agreeable desire to sleep. In some instances, however, he does not feel better, but suffers from præcordial distress which soon passes off. In persons subject to gout and rheumatism the joints may become painful and even swollen, usually returning, however, to normal in a few days. The symptoms in some cases may persist longer.

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* Recent Improvements in the Treatment of Chronic Heart Disease by Exercises and Carbonated Bran-baths. Thomas B. Satterthwaite, Medical Times, N. Y., April, 1898.

The effects of the bath are decided. The pulse is diminished in frequency, its force and volume are increased, the area of dilatation is lessened and the apex approaches nearer its normal position. Thus it will be observed that the exercises and the baths are attended by the same results. Favorable as these results are, they, unfortunately, are not permanent ; hence a long series of baths and exercises is necessary. In this connection Broadbent very opportunely observes, "Too much importance is attached by the advocates of the Schott treatment to the percussing out the area of cardiac dulness and to the diminution it is said to undergo after each bath, more especially when the so-called auscultatory method is employed. This method lends itself very much to the imagination and is absolutely untrustworthy. A shifting upward of the apex-beat is of importance, but it is probable that the diminution of percussion dulness is not due so much to fluctuation in the size of the heart as to encroachment by the lungs on the cardiac area, due to the deeper respirations taken while the patient is in the tub. Accurate delineation of the outline of the heart by percussion is in many instances impossible,—so that a diagnosis of cardiac dulness should not be made by percussion alone."*

Schott explains the action of the baths as due to tissue change produced by a greater power of the cells for the taking up of oxygen. He thus accounts for the need of rest and sleep after the administration of the bath. He holds that there is a reflex stimulating action on the heart which causes more complete and favorable contractions, and, as a result of this influence, hypertrophy. He also calls attention to the possibility of there being direct stimulation of the arteries by the passage of the gas through the skin.

An explanation offered by Broadbent is that there is probably a physiological dilatation of the integumentary capillaries which diminishes the resistance of the blood, thus enabling the left ventricle to complete more thoroughly its systole, and in this way permitting the venous blood to flow more rapidly toward the arterial system. Broadbent admits that the objection to this hypothesis is the slowing of the pulse which takes place during the bath, inasmuch as the lessened peripheral resistance might be naturally looked upon as a means of accelerating the pulse-rate rather than the contrary. He therefore suggests that the slower pulse-rate is due to reflex stimulation.

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* Heart Disease, William H. Broadbent, M.D., London, 1897.

Exercise and baths each accomplish the same results, but the most rapid effects are obtained when they are employed conjointly; they are, however, only useful up to a certain point, after which they are harmful. When used to excess or injudiciously, restlessness, insomnia, loss of appetite and of the general strength, with an irritable and excited state of the nervous-system, are likely to follow.

The general indications for the Schott or Nauheim treatment by exercises and baths are weakened and feebly maintained conditions of compensation, dilatation from depressing diseases, where it is of especial service, and functional neurotic disturbances. In mitral stenosis with feeble compensation, and when obstruction is such that the action of digitalis on the right ventricle would be unfavorable, it will be of signal benefit.

Contraindications are cardio-vascular affections associated with arterio-sclerosis, decided degeneration of the myocardium and the presence of aneurysm. In aortic lesions on account of the danger of syncope it is not to be employed unless compensation is breaking down, when it will accomplish good results.

Venesection.—As a therapeutic measure in conditions of extreme venous engorgement, this is advocated by certain modern observers, when all other measures fail. Although never having resorted to this expediency, its high endorsement by eminent specialists in diseases of the heart makes it seem advisable to present the subject so that the reader may judge for himself.

The indications for its application are conditions of profound venous engorgement when the cavities of the right side of the heart are over distended and almost paralyzed, regurgitation of the blood-current through the lungs and consequently a minimum supply for distribution through the arterial circulation, greatly enlarged liver unless there is cirrhosis, the heart action feeble, its sounds indistinct, the pulse small, irregular and weak, the countenance pale and bathed in cold perspiration, the extremities blue and cold, cyanosis and orthopnœa marked and death imminent. Under these conditions venesection has been employed with success; the withdrawal of from eight to sixteen ounces of blood will reduce the over-distension of the right side of the heart so that it is enabled to act and once more propel the blood through the lungs.

It is a matter of utmost importance to know the condition of the right ventricle before resorting to venesection. If it has undergone degeneration it may be so weak that this measure will not be followed by success. A strong right ventricular impulse should be felt before resorting to venesection. As a substitute, relief may be obtained by applying eight or ten leeches over the epigastrium.

After the abstraction of the blood it is recommended that the liver be relieved by purgatives, when digitalis and alcoholic stimulants should be given.

Drugs.—The administration of remedies which act upon the heart naturally occupies the most important position in the therapeutics of cardiac affections, the chief end of treatment being to induce a more rapid flow of venous blood into the arterial system and to thus establish and maintain the equilibrium of the circulation.

The drugs which are commonly employed in the treatment of diseases of the heart may, for the convenience of discussion, be divided into two classes, viz.: Drugs of primary therapeutic importance, comprising those whose action and application is directed principally toward the heart, together with those whose sphere of action is such that they not only profoundly affect the heart, but in addition possess a much wider range of application. Second, drugs of secondary therapeutic importance, namely, those whose application in cardiac diseases is secondary and incident to their more general use.

I. DRUGS OF PRIMARY THERAPEUTIC IMPORTANCE IN THE TREATMENT OF CARDIAC AFFECTIONS.

Digitalis on account of its prominence in the treatment of diseases of the heart naturally calls for discussion first. Allied to it in action are strophanthus, spartein, convallaria, adonis, oleander and caffein, comprising with a few other remedies of lesser importance what is called the digitalis group. Digitalis, however, occupies the first rank, the others being of secondary therapeutic value, and in some instances but little used.

The action of digitalis is principally upon the cardiac ganglia and heart muscle. In small and moderate doses it induces contractions, invigorates the systole and increases the length of diastole by its influence upon the vagi. The result is a more abundant flow of blood into the ventricles, while the strengthened ventricular contractions propel more blood into the arteries. It also slows the heart and causes it to beat more regularly. The pressure of the blood in the arterial system is raised, while the walls of the arteries and arterioles have a tendency to contract. A dilated heart while under the influence of digitalis becomes smaller. The drug is rather a remedy for a dilated heart than a weak heart. It likewise acts as a hæmostatic. Its action on the kidneys is that of a diuretic, probably on account of the increased blood-pressure of the glomeruli stimulating the kidneys to action. Toxic doses cause a quick, irregular, feeble pulse, rapid respiration, increased vascular tension, sometimes emesis, coma and convulsions. The heart is arrested in systole from paralysis of the motor ganglia, and on post-mortem is found in a state of contraction, which, according to Hughes, is a post-mortem change, as shown by the change of the muscle juice from alkaline to acid.

The sphere of action of digitalis embraces all forms of cardiac disease, but more especially valvular affections with dilatation, dropsy of cardiac origin, pericarditis with effusion, hydrothorax, emphysema and all varieties of weakness of the heart walls. It is claimed by some that when administered for some days in full doses with little or no effect, toxic symptoms may suddenly appear, constituting what is known as the cumulative action of the remedy; others deny that the drug is cumulative, claiming that poisonous symptoms only appear when the effect of one large dose has not passed off before another is administered; a condition which would apply to many other remedies. It is, nevertheless, the opinion of many that a cumulative action is a characteristic of digitalis.

Digitalis is contraindicated in simple or compensative hypertrophy, in some cases of fatty degeneration of the heart, apoplexy, aneurysm and all affections characterized by a high arterial tension. In atheroma it should only be administered temporarily.

The preparations and dosage are matters of importance. The tincture, the infusion, the powdered leaves and the alkaloid are the forms in general use, preference being generally given to the tincture.

The question of the dosage is one in reference to which there is some diversity of opinion. This is doubtless due to a certain degree to the fact that some persons display idiosyncrasies in regard to its toleration. The physiological dose is a few drops of the tincture to twenty; the usual dose is from three to five drops. As a rule, in broken compensation the best effects are obtained from the latter. In some instances, on the other hand, notwithstanding its apparent indication, these doses are attended with a sense of pulsation and fulness, and are not followed by beneficial effects. Under these conditions the remedy should be replaced by another or the dose reduced, giving a teaspoonful of a mixture of ten drops of the tincture in half a glass (℥ iv) of water, or five drops of the first decimal dilution. These doses are especially suitable for delicate persons and children, and for milder forms of disturbance. In emergencies large doses will be required. Jousset, of Paris, gives the remedy as follows: From 20 centigrammes to 1 gramme of the first decimal trituration, that is from 2 to 10 centigrammes of the substance is placed in 125 grammes of water; this amount is given in the course of a day.

When administering digitalis rapid slowing of the pulse should be avoided. Such a change is an indication for its discontinuance or reduction of the dose.

Digitalis in the form of the infusion is preferred when there is much œdema. It is less liable to cause gastric disturbance than the tincture. A drachm every two hours will generally prove effective and is well tolerated. Half-ounce doses every four to eight hours are advised by some. It is important that the infusion should be freshly prepared. This is sometimes difficult to accomplish and is probably one reason why the tincture is more frequently used.

The dropsical conditions where digitalis will act well are those where the pulse is weak, soft and intermittent and the limbs easily pit; when the pulse is hard and the limbs tense and brawny it is ineffectual. Under these conditions the blood-pressure must be lowered by purgation by mercury, and some of the fluid thus withdrawn before the remedy is administered. Digitalis is often effectually administered in connection with other cardiac remedies of its class, notably strophanthus and caffein.

When the stomach cannot tolerate either the tincture or infusion, the powdered leaves may be substituted in the form of a pill, or the alkaloid, digitaline, may be used. Care is necessary in regard to the selection of the latter as some of its preparations are unreliable, and its use is for this reason often avoided. Merck's or Nativelle's digitaline in doses of $\frac{1}{250}$ to $\frac{1}{60}$ grain will prove effective.

Dr. Theodor Schott recommends digitaline subcutaneously or by the rectum in cases where it is not well tolerated by the stomach.*

The toxic effects of digitalis are præcordial oppression, slowness of the pulse, doubling of the heart-beat with only one beat at the wrist, and diminution of the urine. The latter is sufficient indication for the withdrawal of the remedy when it is being administered in physiological doses.

Strophanthus has the same general indications as digitalis, but with some differences. Observers, however, are not fully agreed as to its action. The most generally accepted conclusions point to it as an invigorator of the heart muscle, lengthening the interval between the contractions and dilating the cavities. It is claimed by some that it dilates the arteries and increases the blood-pressure, but others deny this action, attributing the increased blood-pressure to more forcible contractions of the heart. At any rate, it does not contract the arteries. In fatal cases of strophanthus poisoning the heart has been found arrested in diastole.

Strophanthus differs from digitalis in that it is less effective in œdema, especially when associated with mitral disease, while it acts better in conditions of dilated senile heart. Goodno recommends it in failing heart of cardio-vascular sclerosis, associated with interstitial nephritis. The remedy is useful in combination with digitalis, and when the latter is not well tolerated or does not act well, and in the intervals of digitalis treatment. It acts more rapidly than digitalis, but its effects do not last so long. It is valuable in shock in persons with weakened hearts, in cases of cardiac dropsy with renal or pulmonary congestion, and pulmonary œdema with irregular heart. Its diuretic action is limited. Dose, ♏j–v of the tincture. Caution is necessary in regard to the preparation as there are many inferior tinctures.

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* Chronic Muscular Diseases of the Heart, Theodor Schott, M.D., Therapeutic Monthly, February, 1902.

Caffeina has been used upon the same indications as digitalis. Its action is more rapid, but of shorter duration. It slows the pulse, raises arterial tension and is a true diuretic, increasing both the solids and the liquid of the urine. It will bring about free diuresis in twenty-four hours. It is therefore very useful in dropsical conditions and as a substitute for digitalis it is often effective. It possesses the additional advantage of being non-irritating to the stomach, but in some patients the full doses which are often necessary to obtain its action may cause cerebral symptoms, which, however, are transitory and disappear upon the withdrawal of the drug. In urgent cases of heart failure its rapid action renders it valuable. Dose, gr. j–v.

Huchard recommends the following formula for hypodermic administration :

℞. Caffein,	4 grammes.
Salicylate of soda,	3 grammes, 10 centigrammes.
Water,	6 grammes.

The dose is up to 40 centigrammes of caffein.

Huchard repeats the injections every three hours. Jousset recommends injections four times in twenty-four hours.*

Sparteina, the alkaloid of scoparius, is a cardiac remedy of efficacy, but is not widely employed. In toxic doses it causes extreme frequency of the pulse, great feebleness of heart action, intense dyspnœa and vertigo. Death, sometimes attended with convulsions, is caused by paralysis of the respiratory centres in the spinal cord (Foster's Applied Therapeutics). Its therapeutic action is that of a stimulant on the heart acting upon the muscle of that organ through the cardiac ganglia. It is less powerful than digitalis, but its effect is more immediate and its action more prolonged. The influence of a single dose of one and a quarter grains is said in some instances to persist for twenty-four hours. In doses of a third to a half grain it reinforces the heart, slows the pulse and increases the blood-pressure and allows the heart to beat more regularly. Its sphere of application lies in the treatment of diseases of the myocardium and valves where speedy effects are desired, in dilatation from valvular disease, and in enfeebled heart from structural lesions. It is also effective when innervation of the organ is much disturbed, and it is thus indicated in functional disturbances arising from excessive fatigue, either mental or physical, and in nervous cardiac disorders, such as irritable heart, painful sensations connected with valvular disease and in tobacco heart. Dose, gr. ⅙–ij.

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* Practice of Medicine, P. Jousset, M.D. Trans. J. Arschaguni, M.D., N.Y., 1901.

Convallaria bears a close resemblance to digitalis in its physiological action, differing in the following particulars: Its action is more strictly limited to the heart; it does not prolong the diastole to such an extent, but it slows the heart action and increases the force of the systole; it is not cumulative, it is less liable to cause gastric irritation, and it is much less active as a diuretic.

Its sphere of usefulness lies in the treatment of valvular diseases attended with irregularity of rhythm, and obstruction where, like digitalis, it tends to augment the flow of the blood from the veins to the arteries; in conditions of passive congestion arising from weakness of the heart, and when the right side is overtaxed, as in pulmonary congestion and emphysema. It is also useful in strain of the heart from any cause, hence in irritable heart it may be employed to advantage. In renal dropsy it is of no service, but in that dependent upon heart disease it is often effective. Dose, ♏iij–v of the fluid extract.

Coronilla, judging from the experiments of Spillmann and Haushalter, as mentioned by Shoemaker, acts similarly to digitalis and may be classed with it. In organic disease of the heart it increases the volume of the pulse, decreases œdema, promotes diuresis and relieves dyspnœa. Its general indications are the same as digitalis. It is especially recommended by those who have tested the powers of the drug in aortic and mitral lesions where it acts beneficially in affording relief to paroxysms of dyspnœa. It is also said to favorably influence the pulse in tachycardia and to remove reflex symptoms. It has no cumulative effect and does not disturb the stomach. Dose, gr. j–viij.

Oleander has a marked action on the heart and may rightly be ranked among remedies of the digitalis group. Under its influence a weak pulse will become stronger, diuresis will occur, constipation will be relieved and dropsical symptoms, in some instances, dissipated. It should not be administered when there is any irritation of the alimentary canal. It is a powerful remedy, but appears to have been but little used. Dose, ♏v–x of the tincture.

Cratægus oxyacantha will prove a useful adjuvant to digitalis. It may be used in the milder forms of failing compensation, but must not be relied upon alone.

Its indications are more or less dilatation, second sound weak, a weak pulse-rate, much accelerated, irregular and intermittent and mitral regurgitation. Dose, ♏viij–xv of the tincture, in association with digitalis three or four times a day.

Adonis vernalis is another remedy of the cardiac tonic group of secondary importance. It also resembles digitalis, but differs from it in being more powerful and rapid in action. It may be administered for months without cumulative effect and is well tolerated. The effects appear to be present only during its administration. It may be employed with benefit in cases where digitalis is not well sustained and in conditions of idiosyncrasy. It regulates and slows the pulse, increases the force of the impulse, diminishes the size of the heart in dilatation and increases the arterial pressure. It is employed in valvular deficiencies attended with enfeebled compensation and in palpitation of functional origin independent of organic disease. The infusion (ʒj to water ℥xij), dose a teaspoonful every two hours in severe cases, every four in milder; the fluid extract, dose ♏j–ij, increasing to ten or fifteen, and the alkaloid, adonidin, are the forms in which it is administered. The latter is preferable; the dose is gr. $\frac{1}{20}$–$\frac{1}{8}$. Large doses produce emesis. In some ininstances it proves constipating.

Amyl nitris, acting through the motor centres of the spinal cord, affects profoundly the heart and entire arterial system, including the capillaries. The most striking symptom produced by its administration is a sense of fulness in the head, especially in the temporal region, which, if the dose is sufficiently large, amounts to severe pain, flushing of the face, increase in frequency and depth of respiration, rapid, irritable heart action without apparent increase in the force of the systole. Toxic doses cause extreme pallor, dilatation of the pupils, weakness of the heart, great general muscular relaxation, irregular, superficial respiration and change of the arterial blood to the appearance of venous. Its action is rapid and evanescent.

Its application in diseases of the heart is indicated in conditions of angiospasm and increased blood-pressure. It is a most important drug for the treatment of the paroxysms of angina pectoris. In cardiac dyspnœa it affords temporary relief, especially when the condition is dependent upon mitral disease.

It is also effective in small doses for the relief of pain and dyspnœa associated with aortic insufficiency. In heart failure, syncope, especially from shock, anæsthesia, particularly from chloroform, it has proved a powerful agent in averting impending death. It is administered almost entirely by inhalation. In cases of fatty heart the dose should be small and cautiously administered.

The dose by inhalation, the usual method of administration, is from three to five minims. One-quarter of a minim may be given internally.

Glonoin (nitro-glycerine or trinitrin) has the same physiological action as amyl nitrite, but is usually administered by the mouth or hypodermically. It is of more therapeutic importance than any of the remedies of the nitrite group, its sphere of utility in cardiac disease being one of great importance. By its power of dilating the arterioles it assists the circulation by lessening the peripheral pressure, thereby supplying more blood to the surface and affording a certain amount of relief to the left ventricle. It is indicated in conditions of high arterial tension and spasmodic contraction, in rigidity of the blood-vessels and in those cases associated with degenerative changes that occur more in advanced life. In cases of excessive hypertrophy of the left ventricle, in aortic regurgitation associated with arterio-sclerosis, in the dyspnœa of cardiac asthma, of weak, dilated hearts and in all conditions associated with faulty nutrition, —notably angina—in sluggish conditions of the kidneys and scanty urine from stasis, with danger of uræmia, nitro-glycerine will prove of service. Dose, ♏j–ij of the second decimal dilution.

Strychnia is the most powerful and active cardiac stimulant. It operates on the heart muscle through the cardiac motor ganglia in the medulla, increasing the contractile power of the heart muscle and contracting the arteries throughout the system. It is effective in all forms of weakness of the heart due to depressed nervous force and dilatation. In many instances it will constitute a valuable adjuvant to other remedies of the cardio-tonic group, especially digitalis. In fatty heart it should be given with care, as it sometimes induces nervousness and insomnia.

Its action as a general systemic tonic is an important characteristic. It is more frequently employed in the later stages of cardiac dilatation. No remedy is its equal in sudden heart failure from any cause. Its administration under these conditions should be hypodermically, the dose being from $\frac{1}{50}$ to $\frac{1}{20}$ of a grain. In cases of heart failure usually $\frac{1}{20}$ of a grain is administered.

Suprarenal extract is used in organic diseases of the heart. Its indications are weak, fluttering, irregular pulse, dilatation of the peripheral vessel. It may also be given in cases of valvular disease with high tension. Its action is directly on the heart muscle and is very rapid. The effect of a dose may be observed within five minutes after administration, and is continued from five minutes to three hours. It has no cumulative effect. Dose, gr. iij of the powder, loosely packed in a gelatin capsule and dissolved in the mouth. Tablets or tightly-packed capsules are less reliable.

Cactus grandiflorus may be properly classified as an important cardiac remedy, but cannot be compared with digitalis in efficacy. Its physiological action on the heart is through the cardiac plexus of the sympathetic system. It shortens the ventricular systole and increases its strength. It does not appear to interfere with the action of the inhibitory nerve, nor to produce marked vaso-motor effects. It raises the arterial tension. Toxic doses cause reduction of the blood-pressure, irregularity of the pulse and arrest of the heart in systole. Death is preceded by clonic and tetanic spasms from over-stimulation of the cord.

The remedy is indicated both in functional and structural conditions. It has proved useful in palpitation of functional origin as well as that associated with organic disease, in relative insufficiency due to degeneration of the heart muscle, in cardiac weakness after protracted illnesses and especially in aortic regurgitation. In mitral stenosis, according to some observers, it is decidedly contraindicated. It is also valuable in an acute inflammation of the heart, hypertrophy, irritability, hyperæsthesia and pseudo-angina.

The guiding symptom which indicates the remedy is a sense of constriction about the chest generally described as if the heart were being grasped in a vice, or as if the chest wall were surrounded by a band.

Alcohol stands pre-eminently first amongst heart stimulants. Its action is safe and rapid. Whiskey and brandy are the usual forms in which it is administered. It is best given in small doses frequently repeated, as its action is thus better sustained. It dilates the arterioles and increases the force of the heart. For speedy action it may be administered hypodermically. Combined with ether it is one of the most effective heart stimulants.

II. Drugs of Secondary Therapeutical Importance in the Treatment of Cardiac Affections.

When the dose is not mentioned the following remedies are recommended in the form of the mother tincture and the 2x and 3x dilutions and triturations. Exceptions are *acidum hydrocyanicum* and *lachesis*, which should be given in the 6x dilution and 6c trituration and upward respectively, and *aurum*, which may be given in the 3x to the 6x trituration, and in the form of the 2x dilution of chloride of gold and soda, of this, five to ten drops in water may be given three times a day.

When tinctures or dilutions are used, from five to ten drops may be mixed in four ounces of water, and a teaspoonful of the same given every hour or two hours, according to the demands of the case.

Acidum hydrocyanicum may be used in palpitation and other functional disturbances, especially in the early periods of hypertrophy and in pseudo-angina. The remedy should be used with caution.

Acidum oxalicum has been proved useful in some forms of angina pectoris. Sharp, shooting pains in the præcordial region, dyspnœa, numbness and weakness of the back, loss of power and coldness of the lower extremities are guiding symptoms.

Aconitum is indicated in the early stages of acute inflammations of the peri- and endocardium attended with fever, restlessness and anxiety. In simple hypertrophy it may be employed with good results to allay the tumultuous action of the heart. In aortic disease it has been used for the relief of palpitation. The dose in these cases should be small, and when the palpitation has subsided the remedy should be withdrawn. Guiding symptoms are sthenic conditions, violent palpitation with anguish, constricting, boring pain and dull pressure in the præcordial region.

Agaracin is well spoken of as having been productive of excellent results in cases of extreme dilatation of the right heart from disease of the mitral valve, from that secondary to pulmonary emphysema, and in cases of heart failure from any cause. Dose, gr. j–iij of the 1x trituration every one to every three hours.

Ammonium carbonas, through its power of temporarily increasing the activity of the heart, is useful in dyspnœa dependent upon dilatation and attended with palpitation and cyanosis, and in catarrhal conditions of the air passages associated with pulmonary congestion. Dose, gr. ij–v.

Antimonium tartaricum, in its sphere of action in catarrhal conditions of the respiratory tract, characterized by much rattling mucus, rapid pulse and oppression, will prove valuable in pulmonary œdema with dilatation of the heart when there is a large amount of mucus in the lungs, great dyspnœa and cyanosis.

Apis mellifica is an effective remedy for the relief of dropsical effusions, especially when the kidneys are implicated, and in the later stages of pericarditis.

Apocynum cannabinum, although especially indicated for ascites depending upon hepatic diseases, is also useful in dropsy of cardiac origin. Dose, *θ*, ♏ij–v.

Arsenicum album exerts a valuable tonic influence in organic heart disease. Its scope of action lies mainly in conditions dependent upon malnutrition, degeneration of the heart walls, with development of neuroses, and in dropsical effusions. Under its administration, dyspnœa, palpitation and œdema improve and an intermittent pulse will become regular. When there is excessive hypertrophy and palpitation, or when the heart is weak and irritable and there is much dyspnœa, arsenic will often afford relief. In anginous conditions attending cardiac lesions, in the latter stages of valvular diseases with dropsy and involvement of the kidney, it is of great value. Violent palpitation, especially at night, with great anguish and anxiety; irritability of the heart, with irregularity of the pulse; pulse quick, small, weak; dyspnœa; præcordial pain and angina pectoris, are special indications pointing to its application.

Arsenici iodidum is indicated in valvular disease associated with degeneration, arterio-sclerosis and in fatty heart, and in all forms of malnutrition. In aortic disease with arterio-sclerosis it will sometimes act better than the other iodides. It possesses the advantage over potassium iodide in not disturbing the stomach. In aortic disease, in the presence of gouty diathesis, it has proved effective.

Asclepias tuberosa acts upon the serous membranes of the heart. Its application seems to be limited to subacute inflammations of the pericardium with effusion, where it has proved of value.

Aurum has the same general indications as iodide of arsenic, and is to be employed in similar conditions, especially in interstitial changes in the heart muscle and walls of the vessels, when of syphilitic or gouty origin.

Barii chloridum, in addition to its action on the glandular system, has a marked effect upon the circulatory system similar to digitalis. Its primary action stimulates the cardiac muscle and increases the blood-pressure. Secondarily it depresses the heart and arrests it in systole. It may be used in forms of cardiac disease in which there is weakness of the heart muscle. Da Costa recommended it in valvular disease as a cardiac tonic wherein it relieves cardiac distress and pain, increases the tone of the vessels and causes diuresis. Hare found that it acted well in functional disturbances.

Belladonna is indicated in acute inflammations of the heart attended with symptoms of acute cerebral congestion, increased force of the pulse, throbbing of the temporal vessels and delirium. Præcordial pain and over-action from strain are relieved by it. In chronic conditions of the heart it has no action.

Potassii bromidum, sodii bromidum, strontii bromidum, while depressing to the heart in large doses, in small act very beneficially in functional disorders, both of neurotic and organic origin. They are valuable remedies for the relief of nervous excitement, palpitation and irritability of the heart in well nourished or sthenic cases. The bromide of soda is the preferable form. In cases where this disagrees with the stomach the bromide of strontium may be used. The latter will afford marked relief in nervous dyspepsia and gastralgia when associated with disease of the heart. Dose, gr. v–x.

Bryonia alba is a remedy of importance in acute rheumatic inflammation of the serous membranes of the heart in the stage of exudation. Passive pain in the præcordial region, stitches, violent rapid action of the heart, pulse full, hard and rapid, are special indications for its use.

Camphora is useful in small doses for palpitation of nervous origin. Dose for heart failure is thirty to sixty drops of a 1-per-cent. solution in olive oil. In the heart failure of pneumonia, influenza and typhoid fever and similar exhausting diseases it is used hypodermically. In febrile conditions, with tendency to failure of the circulation, and in exanthemata when the eruption does not appear it acts beneficially.

Cantharis is of service in violent inflammations of the serous membranes of the heart, especially pericarditis, during the stage of exudation. Pulse full and irregular with tendency to syncope.

Cimicifuga has a decided, but much less marked action on the heart resembling digitalis. It is useful in rheumatic peri- and endocarditis, more particularly when occurring in women with suppression of menses, intense frontal headache, delirium and pain in the left inframammary region and down the left arm. It may also be employed in irritable and feeble conditions of the heart, especially when associated with chorea, and in palpitation connected with uterine disorder. It has been used by Hale in angina pectoris, presumably in severe pseudo forms. In fatty heart it may prove of more service than digitalis.

Coffea will prove useful, on account of its mild stimulating effect, in conditions of feebleness of the circulation. Its action is more marked in those unaccustomed to its use. It should be remembered when mild stimulation is required, especially in the case of children.

Colchicum is effective in acute inflammations of the serous membranes of the heart, of gouty or rheumatic origin, especially the former. It acts better in pericarditis than in endocarditis and is a cardinal remedy for the affections of gouty subjects and in metastasis of gout to the heart. It is also recommended in pericarditis of subacute and chronic forms and in hydropericardium. Severe pain in the region of the heart of a boring nature, anxiety, oppression, sensitiveness to pressure, violent palpitation, irregular or hard, full pulse point to its selection. The alkaloid colchicine is generally to be preferred to the drug itself. Dose of the latter gr. $\frac{1}{100}$–$\frac{1}{50}$.

Cupri arsentis, it is claimed, acts best in certain anomalous disorders of the heart of a functional character, marked by arrhythmia, heart action at one time strong, at another feeble, appearing in paroxysms between which physical examination will fail to reveal any abnormal features; conditions, in short, which have been termed cardiac chorea.

Collinsonia may be employed with satisfactory results in purely functional disorders of the heart of reflex origin, characterized by rapid, weak action or excessive action without force. In irregular action of hypertrophy it has also proved of decided benefit. In heart symptoms of reflex origin its influence on the pelvic organs should be borne in mind.

Ether is indicated for sudden heart failure. It is liable to produce an abscess at the point of injection, but the objection is of small account in view of an emergency. Dose, ♏xx–xxx hypodermically.

Erythrophleum or *casca cortex*, although little employed, according to Germain Sée and Brunton, is a useful remedy in certain conditions of the heart. The powder when inhaled causes most violent and prolonged sneezing, hence caution is necessary in its preparation. In excessive doses the drug produces most marked muscular relaxation. Poisonous doses in animals cause vomiting, slow, irregular heart, quick, labored respiration, contraction of pupils followed by sudden dilatation, convulsions and death, consciousness being retained up to the time of dissolution. In medicinal doses it strengthens the heart, slows the pulse, contracts the arterioles and acts as a diuretic by contracting the arteries and increasing blood-pressure. It has been considered useful, according to Brunton, in dilatation without valvular disease, in mitral disease and dropsy. Germain Sée recommended it in dyspnœa. Drummond considers casca bark preferable to digitalis when an action is desired upon the peripheral inhibitory nerves of the heart (Hale). It is more liable to cause gastric disturbance than digitalis. Dose, gr. v–x.

Potassii ferro-cyanuretum may be employed, according to Hale, for those cases which appear to require both digitalis and iron. It has been given for hypertrophy and in functional disorders. Palpitation, ringing in the ears, vertigo, intermittent pulse, sense of suffocation, and disturbed sleep are amongst the nervous phenomena indicating its use. Care should be taken not to confound the remedy with cyanide of potassium.

Ferrum, in cardiac affections, has a valuable effect by improving the general nutrition. In fatty degeneration, dilatation and valvular disease, it will tend to maintain compensation by enriching the blood. In murmurs of anæmic origin it is usually very decidedly indicated. The tincture of the chloride in the usual doses is the most effective form in which to administer the remedy in these conditions. Iron may also be employed in another way, namely, in the form of the triturate, not lower than the 3x, when it will be found useful in cases of hypertrophy with accelerated action of the heart, rush of blood to the head, throbbing headache, tendency to hyperæmia of the brain and lungs, with ischæmia of the peripheral parts.

Gelsemium may be employed for a limited period in the first stage of acute inflammation of the heart in non-rheumatic cases, rarely if ever in rheumatic, and never in those associated with renal disease. It will at times prove of service in hypertrophy to relieve the forcible pulsation, being indicated by conditions of cerebral congestion and tendency to mental torpor. Palpitation of tobacco heart is relieved by it.

Iberis possesses decided influence over the heart, but does not appear to have gained much reputation as a cardiac remedy. It controls excessive action and softens a hard pulse. Its sphere of usefulness lies in hypertrophy with increased action with a corresponding increase of force. Diminution in the urine, bronchial irritation and deficient action of the liver as evidenced by clay-colored stools are additional indications.

Ignatia is limited in its use in cardiac disease to various nervous and functional disturbances. In palpitation of reflex or neurotic origin and in that associated with hypertrophy, it will prove effective, especially in hysterical patients. A sensation of sinking in the epigastrium, and of great weakness point to its use in such cases.

Potassii iodidum, sodii iodidum, strontii iodidum are important remedies for retarding the progress of the morbid processes in all forms of sclerosis and degeneration, both non-specific as well as syphilitic. In aortic disease, angina (during the intervals of the attacks), chronic myocarditis and fatty degeneration, they should be given for a long period in moderate doses (five to ten grains in plenty of water three times a day).

Kalmia latifolia possesses marked action upon the heart, and is indicated by the presence of sharp, stabbing, shooting pains, dyspnœa, and slow, weak pulse. It is also useful in the palpitation of hypertrophy and in the milder forms of angina and rheumatic endocarditis.

Kola acts beneficially in functional heart disturbances, palpitation, arrhythmia, etc., especially from gastric irritation and fatigue. It has also been employed as a general tonic for the heart.

Lachesis acts on the heart, affecting principally the endocardium rather than the pericardium. It is considered valuable by some observers in endocarditis of a malignant type in association with pyæmia and typhus, and in certain nervous symptoms depending upon organic diseases, viz.: Trembling, irritability of the heart after damage from acute inflammations and irritation of the glosso-pharyngeal nerve causing a sensation of choking, or irritation of the throat, or a sensation as if anything touching the throat could not be borne.

Laurocerasus is employed in nervous and organic affections of the heart characterized by spasmodic contraction of the throat and chest, inducing paroxysms of suffocation and great nervous excitement.

Lilium tigrinum is of value in nervous affections of the heart depending upon valvular or uterine disturbances. Special symptoms pointing to its use are the presence of a dull pain in the præcordial region, or a sensation of a heavy weight over the heart, or sharp pains or sensations as if the heart was being firmly grasped and relaxed alternately. Attending these symptoms there is palpitation for which condition it is of decided benefit. It is also of service in pseudo-angina of hysterical or reflex origin.

Lobelia, while acting as a depressant to the heart in full doses, in minute doses will stimulate that organ, and is indicated in functional disorders characterized by severe præcordial oppression, feeble circulation, weak, irregular, empty pulse; sinking sensation in the epigastrium, anxiety, apprehension of death, cold face and extremities, cough and cardiac neuralgia. It has proved of benefit in asthma with hypertrophy, pseudo-angina and cardiac chorea.

Lycopus will prove of service in conditions of irritability and weakness of the heart from rheumatism, debility, neuroses, or from the effects of tobacco. It relieves palpitation of nervous irritation or organic disease, rendering the pulse slower, fuller and more regular. Tenderness and constricting pain, rheumatic aching and acute darting pains about the heart, oppression and distress with irregular intermittent and feeble pulse or pulse quickened at each respiration and extremely variable, are among its characteristic indications. Dose, *θ*, ♏j–v.

Morphia in heart disease occupies an important place. In the later stages of mitral stenosis and insufficiency with all the evidences of general venous congestion, when the heart is active and even tumultuous in its efforts to overcome the obstruction to the circulation, and the patient is slowly becoming asphyxiated, it will afford relief. Any narcotic action should be avoided. The effect in some instances is decided. The cyanosis lessens, the breathing becomes easier, the heart more quiet and the patient falls into a refreshing slumber. The best results are obtained in mitral disease, yet it may also be used in aortic affections with similar symptoms.

In angina pectoris, when the pain is prolonged and intense, morphine will sometimes act when other remedies fail. The vaso-motor dilators, however, should be tried first. In cases which have been subject to repeated attacks of angina there may be absence of arterial tension and spasm. Here the nitrites are not indicated and morphine should be preferred. Dose, gr. ⅛–¼.

Moschus is one of the most effective remedies for hysterical conditions and nervous palpitations.

Naja acts similarly to lachesis, and is prescribed in the same general conditions. The presence of sympathetic, irritating cough in functional heart trouble points to its use.

Nux moschata is of benefit in functional disturbances with hysterical symptoms or reflex ovarian and uterine origin, marked by the presence of a sensation of faintness, trembling of the heart and spasmodic stitches in the præcordium.

Nux vomica, through the spinal cord, the motor and sensory centres of the brain, affects profoundly the entire muscular system. It is valuable in various gastro-intestinal conditions associated with cardiac disease, and in strengthening the heart muscle in dilatation; but its chief application is through its great alkaloid, strychnine.

Plumbum acts beneficially in retarding the growth of hypertrophy and in atheroma.

Prunus virginiana has been proved of benefit in palpitation of nervous and reflex gastric origin, mitral insufficiency and dilatation with chronic bronchitis.

Rhus toxicodendron may prove of service in rheumatic conditions of the heart characterized by stitches in the præcordial region, painful stiffness of the left arm and other parts of the body.

Spigelia is an important remedy in rheumatic endocarditis and pericarditis, but is more effective in the latter. Pains of a lancinating character which tend to radiate, and sensation of great pressure in the region of the heart with violent palpitation, indicate its use in acute rheumatic inflammation of the heart. It may also be used in nervous palpitation, with neuralgic pains of a similar character.

Spongia has been used for paroxysms of palpitation, pain, dyspnœa and great anxiety; pulse full, hard and frequent.

Squilla, or *scilla maritima*, on account of its action on the urinary tract, is of great value for the relief of dropsy of cardiac disease. In weak heart with dropsy or bronchial catarrh it is useful. Its diuretic action is increased by the addition of digitalis and calomel. One of the active principles, scillotoxin, is, according to Brunton, a cardiac tonic. Medicinal doses slow the heart and raises arterial tension.

Stigmata maydis acts as a cardiac stimulant and diuretic. It renders the heart action slower and strong, regulates the rhythm and causes almost immediate increased activity of the kidneys (Hale). It has proved of service in organic heart disease with dropsical effusions and in decreased amount of urine. It has no unpleasant after-effects. Dose, ♏xx–lx of the fluid extract three times a day.

Tabacum, in the form of trituration and dilution, has been employed in persons not accustomed to its use for nervous affections of the heart characterized by irregular pulse and nausea. Pain around the heart, anginal in character, radiating from the sternum to the left arm, may be relieved by it.

Veratrum viride is of importance in over-active hypertrophy and irritable heart. It is contraindicated in dilatation, valvular disease, fatty degeneration and weakness of the heart walls. In the early stages of acute inflammations with high fever, when the restlessness and anxiety which usually indicate aconite, is absent, veratrum viride will act well if administered in small doses. Sthenic conditions with forcible cardiac action, cerebral and pulmonary congestion, are its general indications.

SECTION II.

DISEASES OF THE PERICARDIUM.

PERICARDITIS.

PERICARDITIS is inflammation of the pericardium. The inflammatory process may involve the parietal or visceral layers and affect one or several portions, or the entire membrane. When circumscribed it is confined to the visceral layer at the base of the heart. The disease may be acute, subacute or chronic, but the line of demarcation between the acute and subacute forms is not always clearly defined. Pericardial inflammation varies in intensity, rapidity of progress, extent of pathological change and termination. There are two general forms of the disease, the fibrinous or dry, a relative term used because the amount of the effusion is small, and the sero-fibrinous, where the effusion is copious. It is also described as purulent, hæmorrhagic and tuberculous, according to the associated pathological changes.

Acute Pericarditis.

Ætiology.—The disease is more frequent in the young and middle-aged. In the great majority of instances it is a secondary affection, arising from the presence of toxic matter in the blood, either excrementitious or infectious. As a primary affection it is very rare, although such cases apparently occur occasionally, especially in young children.

Rheumatism is the most frequent factor in its causation. It is variously estimated that from 50 to 70 per cent. of all cases arise secondarily in the course of acute rheumatic arthritis, where it usually appears in the first or second week of the disease, though it may develop later. The greater the intensity of the rheumatism the greater the liability of complications, yet intensity of the rheumatic inflammation is not necessarily a measure of the liability of implication of the pericardium, as pericarditis may occur in the milder as well as the severer form of rheumatism. A rheumatic inflammatory process may sometimes first manifest itself in the serous membranes of the heart. In such instances the arthritic symptoms may appear later and only to a moderate degree. The predominating symptoms under these conditions will be those referable to the heart.

Pericarditis may also be associated with acute tonsillitis in rheumatic subjects.

Bright's disease is next in frequency after rheumatism. The serous membranes of the heart are prone to inflammation in renal disease, especially in persons over fifty years of age. This tendency varies according to the type of nephritis, being more frequent in the chronic interstitial variety. It occurs late in the course of the disease and is an unfavorable indication. Its onset may be so obscure that physical signs alone will reveal its presence. The liability to pericarditis in connection with Bright's disease is greater in cold and changeable climates than in those which are mild and equable. Gout may also give rise to pericarditis, which may appear during the course of the affection as a terminal complication.

Eruptive fevers sometimes act as causes, especially in children, although the tendency is not strongly marked. As a complication of diseases of this class, pericarditis is most frequent with scarlet fever, being associated with measles, typhoid, typhus and smallpox in rare instances only.

Extension of inflammation from neighboring structures, notably the pleuræ and lungs, may involve the pericardium and constitute an important causal factor.

Tuberculosis is not an uncommon cause. The pericardium may become involved as a part of a general tuberculosis, or the disease may arise secondarily from the lungs, or from infection through the mediastinal glands. Cases regarded as primary, at the autopsy have frequently proved to be of tuberculous origin.

Pericarditis also occurs as a complication in acute alcoholism, of myocarditis, phlebitis, pyæmia and septicæmia. It may likewise be associated with disease of the aorta, bronchial glands and œsophagus, with aneurysm, cancer and morbid growths in the vicinity. Finally, it may arise from traumatism.

The presence of staphylococci, pneumococci and bacilli coli has been demonstrated as existing in pericardial inflammation, but a distinctive specific micro-organism has not been discovered.

There are two forms of acute pericarditis: the fibrinous and sero-fibrinous.

1. Fibrinous Pericarditis.

SYNONYMS.—*Plastic Pericarditis; Dry Pericarditis.*

This is the mildest and, at the same time, the most common form of the disease. It is characterized by a fibrinous exudation, the effusion which is present being scarcely appreciable.

Morbid Anatomy.—The morbid changes may be localized or general. The former is more frequent. The initial phenomenon is hyperæmia of the serous membrane which becomes swollen and injected, disclosing ecchymotic points over its surface. This is followed shortly by a deposit of fibrin which gives the surface a rough, grayish appearance. In severe forms the fibrinous deposit becomes more dense, and the natural movements of the pericardial surfaces, one over the other, causes the exudation to assume a honey-combed appearance, grayish-yellow in color. In the later stages the exudation becomes partly organized and, as the result of the constant action of the heart, the pericardium assumes a villous appearance, *cor villosum*, described by ancient writers as "hairy heart." Effusion of serum is always present, but small in amount, hence the inflammation is called dry, in contradistinction to the sero-fibrinous form. Frequently myocarditis also develops.

Symptoms.—As a secondary affection its early recognition is often more or less obscured by the symptoms of the primary disease; moreover, in many instances, the subjective symptoms of pericardial inflammation are sometimes absent, though the lesion is present; especially is this liable to occur when the pericardium becomes involved as a complication of acute articular rheumatism.

It is only in the more severe forms that symptoms pointing to the heart are recognized. There may be a sensation of distress and constriction referable to the heart, tenderness on pressure and a dull pain. Pain is usually more or less marked in the early stages. It may extend over the sternum and down the left arm and back, and sometimes as far as the abdomen.

The pulse is accelerated, and there may be palpitation and some dyspnœa. The temperature rarely exceeds 102° or 102.5° F. Severer forms are occasionally encountered with corresponding intensity of symptoms. The urinary symptoms will depend upon the ætiology; in most instances the urine is high colored and acid.

PHYSICAL SIGNS.—*Inspection* shows an increased vigor of the apex-beat. *Percussion* is negative. *Palpation* may occasionally disclose the presence of fremitus in the early and later stages, when the membranes are least moist. It is usually most marked over the right ventricle. *Auscultation* will reveal a double friction or "to and fro" murmur over a limited area of the præcordial region. This sound is characteristic, though it should not be relied upon alone for diagnosis. It is caused by the inflamed and roughened pericardial surfaces rubbing one against the other, and may usually be heard with maximum intensity in the fourth and fifth interspace, and adjacent part of the sternum. Another point where it may be heard is at the juncture of the heart and aorta. A pericardial murmur is audible as a rule only over a limited area, though occasionally it is diffuse, being discernible over the whole præcordial space. One of its chief characteristics is its superficial nature, the ear readily detecting its nearness to the surface. Mild pressure with the phonendescope or stethescope will increase its intensity, while forcible pressure will cause it to disappear. The sound is also influenced by position and respiration. The quality will exhibit great variety, being soft, rubbing, grating or creaking, the rubbing and grating sounds being most frequently observed. The creaking sound may be heard in the later stages. The murmurs are generally double, and although produced by the action of the heart, they are not always exactly synchronous with its rhythm, usually exceeding the heart-sounds in duration.

It is well to have in mind in this connection that a pleuro-pericardial friction murmur may be associated with an endo-pericarditis, particularly in pneumonia. It may also occur in phthisis. This sound is heard over the left border of the heart and is characterized by being much influenced by respiratory movements. Holding the breath or deep inspiration may cause it to disappear. It is observed as a combination of the "to and fro" pericardial sound with a respiratory murmur. It is usually intensified by inspiration and lessened by expiration. In phthisis a loud, systolic click may be heard, due to compression of a thin layer of lung and the expulsion of a bubble of air from a small bronchus or from a softening point.

Diagnosis.—While the disease may be overlooked, it is not readily confounded with other lesions of the heart. The "to and fro" rubbing sounds with their superficial nature may be regarded as diagnostic, although not absolutely so, as excessive dryness of the pericardial surfaces and calcification of the coronary arteries may produce friction murmurs.

Endocardial murmurs can be distinguished from the pericardial by their blowing qualities. They are more constant and distinct, each sound has its own area of intensity and direction of transmission beyond the region of the heart itself. Double aortic lesions it is stated may simulate the pericardial rub, but the transmission of the murmur and the attending symptoms will readily enable a distinction to be made. Plastic pericarditis may be differentiated from the serous form of the affection by the distinctive physical signs and symptoms which characterize the latter.

Prognosis.—This is generally favorable, but in cases characterized by a low state of the system, as in nephritis, it is grave. Complete resolution is probably rare, as connective tissue formations are liable to result in adhesions. These, however, are not necessarily serious, though they may lay the foundation for future trouble. Unfavorable cases may be followed by effusion and the disease thus constitute the first stage of sero-fibrinous pericarditis. The affection may also assume a chronic form in which case it is generally tubercular with extensive thickening of the pericardial membrane.

2. Sero-Fibrinous Pericarditis.

SYNONYM.—*Pericarditis with Effusion.*

Sero-fibrinous pericarditis is the most serious form of pericardial inflammation. It is an inflammatory process of the pericardium characterized by a fibrinous exudation and a more or less abundant serous effusion into the pericardial sac. It is divided anatomically and clinically into three stages.

Morbid Anatomy.—The pathological changes may be grouped under three heads representing the three stages of the disease, according to which the morbid conditions vary. The first stage is attended with hyperæmia, roughness, detachment of the epithelia and deposition of inflammatory lymph. The phenomena encountered at this stage of acute sero-fibrinous pericarditis correspond to the conditions as they occur in the plastic or fibrinous variety of the disease, except that the changes are more pronounced. In proportion to the amount of fibrin present the pericardial membrane is thickened, attaining in some instances a thickness of a quarter of an inch or more. In appearance it is rough and shaggy. The second stage is characterized by an effusion of clear straw-colored serous liquid in which flakes of fibrin are floating. The effusion will vary in amount from two to twelve ounces; occasionally it is much larger. It usually first appears at the origin of the great vessels as they arise from the base of the heart. The fluid accumulates around the heart and as it increases in quantity lifts the organ and forces its way to the bottom of the pericardial sac. The third stage is that of absorption. The more fluid portions of the exudation will become absorbed, while the more fibrinous remain and give origin to adhesions between the visceral and parietal layers of the pericardium. The union between the two surfaces is very firm, causing obliteration of the pericardial sac to a greater or less extent. The exudation may sometimes become purulent, giving rise to the purulent form of the disease. Again, sometimes the serum may remain and the condition become chronic.

The influence of these changes upon the heart muscle will depend upon the extent and intensity of the inflammatory process. In most instances there is a moderate degree of myocarditis. In protracted cases the changes are more pronounced, the myocardium becomes sodden and softened and the heart dilated. The effect of adhesions tend to the causation of hypertrophy, which in time may lead to dilatation and degeneration of the heart muscle.

The micro-organisms found in association with sero-fibrinous pericarditis are commonly the streptococcus, the staphylococcus and the pneumococcus.

Symptoms.—The course of the disease is also divided into three stages, corresponding to the pathological changes. Its clinical manifestations, however, are characterized by contrasting variations.

As a complication sero-fibrinous pericarditis may fail to elicit recognition, sometimes even physical signs being negative. This is especially true when it occurs with pleurisy, pneumonia or other intrathoracic diseases. Its onset is also liable to be insidious, especially in the young, and when in association with nephritis and tuberculosis. On the other hand, it may be ushered in with a chill.

Disturbances of the circulation appear early in the course of the disease, and not infrequently changes in the pulse will be the first indication of its approach. The pulse will become over-full and strong, accompanied by a tumultuous heart action, of which the patient will be conscious.

The characteristic earlier symptoms of sero-fibrinous pericarditis are pain and tenderness in the præcordial region, palpitation, dyspnœa and disturbances of the circulation.

Pain in the region of the heart is frequently, although not always, associated with the initial symptoms, disappearing or moderating when effusion occurs. It is usually of a dull, aching character and often not severe, yet it may, on the other hand, be lancinating, constrictive and the source of intense suffering, even simulating angina pectoris. It is most intense below the nipple and over the lower third of the sternum ; from this region it may radiate over the chest to the epigastrium, left hypochondrium and down the arm. In rare instances it is confined to the back. It may be constant or only present on deep inspiration or pressure. There will be more or less tenderness, while pressure over the epigastrium upward beneath the cartilage of the ribs will cause pain. Peripheral pains are a feature in some cases.

Fever is present, but it is not high, usually remaining below 102.5° F. When the primary disease is attended with elevation of temperature the onset of the pericardial complication may cause an increase of the fever. An exacerbation of fever, therefore, in the course of a disease likely to excite cardiac complications should always direct attention to the heart, especially in acute articular rheumatism, when the rise of temperature is not accompanied with indications of the invasion of additional joints.

As the second stage is entered and the effusion presses upon the heart, corresponding changes in the circulatory symptoms ensue. If the effusion is not large, the apex-beat may become feeble and almost imperceptible, while the pulse remains full and strong. This is an important diagnostic point when taken in connection with other symptoms. On the other hand, if the effusion is excessive, the radial pulse may become small, feeble and intermittent, and may even disappear during inspiration (*pulsus paradoxus*). The effect of emotional influences will generally be very marked, and constitute a feature of the disease.

Accompanying the circulatory phenomena of the second stage and like it, dependent largely upon the effusion, there will be dyspnœa; in some cases this may be excessive; the countenance will be livid and expressive of the keenest suffering. Rest will be difficult in any posture, the left dorsal decubitus being generally preferred, as it impedes the heart less. When the exudation is very abundant the surrounding structures will be affected. The left lung may become obstructed and congested from the pressure of the distended pericardial sac, which may also produce obstruction of the trachea and œsophagus, adding to the difficulty in breathing and causing dysphagia. In exceptional cases the pressure on the recurrent laryngeal nerve may cause loss of voice. The heart action may become so feeble that the circulation will be impeded, cyanosis, distention of the cervical veins and dropsy develop, and danger from sudden death become imminent. Patients suffering from pronounced types of pericarditis are inclined to be restless, but movement may be restricted by the accompanying rheumatic arthritis. Headache, insomnia and slight delirium are not uncommon. Vomiting is sometimes a prominent symptom, and is generally regarded as of nervous origin. Some patients are intensely restless and irritable, and in rare instances serious nervous phenomena develop. There may be delirium, active, noisy and even violently maniacal, especially at night, or the delirium may be low and muttering. Sometimes there is a transition from one type to another. At times the condition may resemble delirium tremens, meningitis and dementia. There may also be subsultus tendinum, jactitation, risus sardonicus, tonic or clonic spasms and coma. Delirium in acute articular rheumatism is often an indication that the serous membranes of the heart have become involved.

When absorption of the effusion commences the patient passes into the third stage, and in favorable cases the symptoms ameliorate and gradually improve until resolution is established.

Physical Signs.—*Inspection* may show the countenance anxious, and later pale and more or less cyanosed. As the disease advances respiration becomes labored and irregular, with diminished movement of the left side. The veins of the neck may be distended and pulsating. When effusion has occurred there will be prominence of the præcordial region with obliteration or bulging of the intercostal spaces. This effect will be more readily observed in the young when the chest walls are yielding, and in adults when the effusion is excessive. Slight elevation of the nipple may occur in consequence of the distention of the chest walls. The diaphragm and liver may be depressed when the fluid is excessive. In cases of contraction of the lung and old pleuritic adhesions, effusion may occur without causing any change in the contour of the chest walls.

Palpation in the first stage will reveal friction fremitus with the impulse of the apex-beat, but as the second stage appears and the effusion begins to exude the impulse will become diffused, feeble or lost. When detectable it will be observed to have been forced upward and to the left in consequence of the displacement of the heart upward and backward. When the apex-beat is lost it can often be found in the sixth intercostal space by causing the patient to sit up and bend the body forward. Myocarditis may cause a weakened, systolic action, and in consequence an earlier disappearance of the cardiac impulse; on the other hand, old pericardial adhesions may retain the apex in contact with the chest walls. Hypertrophy will have a similar effect. Sometimes a friction fremitus may exist, notwithstanding the effusion, and when absorption has taken place it may simulate dry or plastic pericarditis.

Percussion, in the earlier stages unless the fibrinous exudation is excessive, shows nothing appreciable, but when effusion has occurred there will be marked increase of dulness, laterally and vertically. At first the increase will appear principally upwards and as the effusion accumulates it spreads laterally. The area of dulness is generally described, roughly speaking, as triangular in shape with the apex near the root of the great vessels and the base upon the diaphragm. It may extend upward to the second or third interspace. Laterally the lines of dulness may pass downwards, the right diverging corresponds with the right border of the sternum and runs to the seventh rib, the left meets the base line at the left anterior axillary line. Later, when the accumulation of fluid is excessive, it may reach from nipple to nipple. Inferiorly it may extend as low as the seventh rib and below the ensiform cartilage, the line of dulness being continuous with that of the liver. Position will effect the area of dulness; changing from side to side alters the level of the fluid. Compression of the lung may lead to alteration of the percussion-note, especially in the axillary line; position may change this sign. The presence of an emphysematous lung covering the sac, or retention of fluid behind the heart, may permit of pericardial effusion without apparent increase of dulness.

Absence of resonance in the fifth right intercostal space, to which attention was called by Rotch, is an important sign. This, however, is not always present, even when the effusion is considerable.

Auscultation is important. In the first stage the characteristic friction murmurs, as described under the plastic variety of the disease, may be heard with their maximum degree of intensity over the right ventricle at the articulation of the fourth and fifth ribs with the sternum. These sounds may be audible in the erect and not in the recumbent position. As the second stage is reached they become more feeble and are finally lost. Coincident with the disappearance of the friction murmur, the heart-sounds will become muffled and indistinct, first disappearing at the apex and last at the aortic and pulmonary regions, until no longer heard. The second sound, however, may remain clearly audible over the extreme base of the organ. Owing to compression of the lung, the vocal resonance and respiratory murmurs will be absent from the area of pericardial dulness, or give place to bronchial or broncho-vascular breathing. As absorption occurs and the disease passes into the third stage, the heart-sounds return.

X-RAY DIAGNOSIS, see Part VII.

Diagnosis.—Diagnosis will rest mainly on the physical signs. The friction murmur and the increased area of percussion dulness with its triangular shape are characteristic features. The early recognition of dulness in the fourth and fifth intercostal space is important. Rigors or chills occurring in the course of acute articular rheumatism, or the exanthematous fevers, should always direct attention to the heart. In the first stage pericarditis may be confused with acute fibrinous pleurisy and endocarditis; in the second, with pleurisy with effusion, dilatation of the heart and hydropericardium. Acute pleurisy of the left side may simulate pericarditis. Irritative cough may occur with both diseases. Pleurisy is distinguished by the pain being of a more acute character, and greatly aggravated by respiratory movements. In pericarditis the pain is not usually so intense, and is not so much influenced by position. Again, the friction murmur of pleurisy is synchronous with the respiratory movements and ceases when respiration is arrested, while the pericardial murmur is distinctly connected with the heart.

Sero-fibrinous pericarditis may be easily distinguished from endocarditis. The endocardial murmurs are markedly regular, while those of pericarditis are changeable in strength and character. The pericardial murmur is the rubbing, scratching sound already described, while the endocardial is of a blowing character; it is further distinguished by its superficial nature and localization, while its intensity is increased or decreased by change of position and pressure. The maximum intensity of pericardial murmurs is over the right ventricle at the articulation of the fourth rib with the sternum. They are not diffused beyond the præcordial region. Endocardial murmurs are diffused to the right and left and transmitted along the arterial trunks.

In the second stage sero-fibrinous pericarditis may be distinguished from pleurisy with effusion from the fact that the latter has its characteristic physical signs which occupy the anterior, lateral and posterior aspect of the chest; thus there will be dulness extending to the left beyond that of pericardial effusion.

If there is dulness posteriorly in pericardial effusion, it may disappear by placing the patient in the knee and elbow position. Moreover in pericarditis the area of percussion dulness will be triangular in shape when there is much effusion, a feature absent in pleurisy.

Encapsulated pleuretic effusions connected with the antero-lateral portion of the chest are difficult to diagnose, especially in the absence of the friction murmur. They may be distinguished by clearness of the heart-sounds and the displacement of the apex-beat a little to the right. In pericarditis, on the other hand, the friction murmur may be heard over the base of the heart, the action of which is disturbed and sounds distant and muffled.

Dilatation of the heart may present difficulties in diagnosis.

The condition has been mistaken for pericardial effusion and the heart tapped. The following are points of differentiation: In dilatation there is a history of chronic disease of the heart. In pericarditis there is a history of acute disease, rheumatism, gout and the exanthemata, or tuberculosis. In dilatation, fever and pain are absent, while these symptoms as a rule appear to a greater or less degree in pericarditis. Again, in dilatation, the apex-beat is usually discernible and palpable, although diffuse, but in pericarditis with effusion it is feeble, and later absent. In pericarditis, præcordial fulness will appear, especially in young subjects. In dilatation the area of dulness will vary with the chambers dilated; it will not extend as high or be influenced by position. In pericarditis the area of dulness is suggestive of a triangle as to shape, while its outline will change with the position. There is also dull tympany in the axillary and subscapular region. In dilatation, auscultation will reveal the first sounds shortened, with no friction sound, and often one or more endocardial murmurs. In pericarditis the sounds are indistinct and there may be a double friction murmur over the base of the heart.

Hydropericardium may be distinguished from pericarditis by the symptoms and condition attending the accumulation of fluid. It frequently follows hydrothorax.

Course.—The disease may pass rapidly through its three different stages or it may be protracted. In the latter instance it is frequently called sub-acute. In favorable cases, especially those arising from rheumatism, it will usually complete its course in two weeks. Relapses and recurrent attacks are not uncommon, while the acute form may pass into the chronic. The rapidity with which the effusion accumulates is extremely variable, it may reach its height in a few hours, or several weeks may elapse during which it slowly exudes. Absorption is equally without rule.

Prognosis.—This is generally favorable, though obviously dependent upon the primary disease, the intensity of inflammation and the condition of the patient. Serous pericarditis arising from rheumatism in many instances terminates favorably, though it is probable in most cases that fibrinous deposits and adhesions remain, yet they are not sufficient to interfere with the functions of the heart. Rheumatic sero-pericarditis with a temperature of 105° F., or with delirium, is unfavorable. Occurring with pneumonia it is a serious complication as it contributes to weaken the heart, though, if the pneumonia terminates favorably, the pericardial inflammation will generally resolve. As a complication of pleurisy it is less serious, associated with Bright's disease it is unfavorable, with the exanthemata, favorable, with pyæmia or scurvy the prognosis is bad.

Symptoms indicating recovery are fall of temperature by lysis, amelioration of dyspnœa and nervous symptoms, and a less full and rapid pulse. Convalescence is further indicated by the absorption of the effusion. Unfavorable symptoms are increased elevation of temperature, aggravation of nervous symptoms, delirium, insomnia and greater difficulty in breathing. Delirium with high fever suggests myocarditis and endocarditis. Rapid accumulation of the effusion is unfavorable.

Complications.—During the course of acute sero-fibrinous pericarditis the inflammatory process may extend from the deeper layer of the pericardium or from the pleuræ, particularly the left, and involve the external surface of the pericardial membrane. This condition is called *Mediastino-Pericarditis or External Pleuro-Pericarditis*, and is most frequently observed in association with tuberculous pleurisy. Various forms have been described, namely, fibrinous, serous, purulent and hæmorrhagic, attended with adhesions, hypertrophy and dilatation. The mediastinal tissue itself is often involved. The essential features in the morbid anatomical changes induced by the implication of the mediastinum as a complication of pericarditis is the presence of a membranous or cord-like adhesion binding the base of the heart and great vessels to the mediastinal structures, sternum, spinal cord and œsophagus. There will also be thickening of the connective tissues around the great vessels, and in some instances the newly formed tissue may produce a considerable amount of constriction of the venous trunks and aorta. There are no positively definite symptoms to mark the appearance of mediastino-pericarditis. Distention of the jugular veins with each forced inspiration and their subsidence on expiration, in connection with the presence of the pulsus paradoxus, may be considered as presumptive evidence of the lesion, but as these phenomena may occur in other conditions they cannot be regarded as positive. An important feature is the detection of the presence of a friction murmur partly cardiac and partly pleuritic. This sound is more distinctly heard along the left border of the heart. Momentary arrests of expiration will suppress the friction murmur and leave the sounds produced by the rhythmical action of the heart. These, however, may be absent. During forced respiration nothing will be heard but a pleuritic rub. In normal respiration the inspiratory movements decrease, while the expiratory movements increase the intensity of the sounds.

Treatment.—During the course of a disease in which pericarditis may arise as a complication, the possibility of such a contingency should always be borne in mind, especially in acute rheumatism. The question of preventive treatment, therefore, naturally presents itself first. In rheumatism the salicylates, colchicine, the application of cold and of blisters, have each been advanced as effective preventives. In regard to the salicylates, notwithstanding their undoubted utility in rheumatism, pericarditis appears as liable to occur under their administration as under any other line of treatment. Colchicine is endorsed by Goodno of Philadelphia, who says that in a series of one hundred and fifty cases of rheumatism, collected from various sources, treated in the earlier stages by this remedy, no pericardial complication occurred. Cold to the præcordium in the form of an icebag or Leiter's coil is effective and is widely endorsed. The continuous application of small blisters over the region of the heart is another method which some writers endorse, especially Caton of Edinburgh. The time-honored alkaline treatment is still regarded by many as one of the best preventives of cardiac complications in rheumatic fever. Like blisters and cold it has the additional advantage of not interfering with any other line of medical treatment that may be deemed advisable to adopt. There can be no doubt, with the exception of the salicylates, that each of these methods are more or less effectual as preventives of pericardial inflammation in rheumatic fever, but to claim superiority for any one would be difficult, inasmuch as, in making statistical comparisons, the fact must be taken into consideration that the relative frequency of pericardial complications arising in connection with acute rheumatism necessarily varies.

The management of each case of pericarditis will be largely influenced by the nature of the disease which is the source of its origin. If it arises in connection with rheumatism, anti-rheumatic treatment must be continued, if due to uræmia, active measures for the elimination of the poison should be instituted. The general line of treatment which has been pursued for the primary disease must be continued in conjunction with such special remedial measures as are indicated. In all cases rest in bed is imperative. It is very important to keep the heart as quiet as possible, therefore physical exertion of every kind must be avoided and any excitement from emotional influences strictly prohibited. Flannels should be worn over the chest so as to avoid exposure, and in some cases it will be well to substitute thin flannel blankets for sheets. The diet should be of the lightest nature, consisting of easily digested, albuminous food. Broths, milk, kumyss and eggs are allowable. Fluids in large quantities should not be allowed, as they tend to increase the arterial tension and delay absorption. Stimulants must be given as the pulse indicates. If the heart shows evidence of failing power, sufficient stimulant should be administered to produce the desired effect and continued as indicated. Aromatic spirits of ammonia will prove useful in addition to alcoholic stimulants. If the cause of the cardiac weakness is due to the presence of the effusion, aspiration should be performed and the fluid slowly removed.

For the relief of pain and soreness hot poultices over the præcordial region will in many instances prove soothing and grateful. The simple expediency of applying a layer of thick flannel over the skin will permit of the application of a very hot poultice without burning, and consequent longer retention of the heat. Cold in the form of the ice-bag or Leiter's coil is preferred by many, and is frequently more acceptable to the patient, but should never be employed if it causes discomfort. It is especially indicated when fever is high and when the disease is secondary to the exanthemata, or has a tendency to assume the purulent form. It may be applied in the first and second stages, but with caution in the latter. Subsidence of fever, indicating that the inflammatory process has reached its maximum intensity and is on the decline, is the signal for its withdrawal.

In addition to their use as prophylactics, blisters are warmly endorsed by many observers as effective in pericarditis in preventing effusion and adhesion, if employed early. A blister three inches square applied over the præcordial space is advised. There is, on the other hand, some diversity of opinion in regard to vesication in pericarditis, some practitioners looking upon it as entirely uncalled for. Internal medication, which is most important, is as follows:

Aconite is of utility in pericarditis of rheumatic origin, in that arising in connection with renal diseases or septic infection it is useless. Its sphere of action is limited to the first stage, or until exudation is complete. The presence of fever, hard, strong, contracted pulse, hot, dry skin, thirst, anxiety of countenance, palpitation and fear of death are its chief indications. It may be given in cases accompanied with pain of various types, as well as those in which this symptom is absent. Some observers condemn aconite as absolutely unworthy of confidence on account of its depressing effect, but it is obvious the dosage was too large. To act well aconite must be given in the form of the tincture, five to ten drops in about four ounces of water (half a glass), and a teaspoonful every hour, or a few drops of the second dilution.

Veratrum viride is applicable to some forms of rheumatic origin. It is indicated when the symptoms are of an intense character, but without the anxiety and restlessness indicating aconite, with violent chill, hard, bounding pulse, cerebral congestion, constant burning pain, oppression, violent, tumultuous heart action, and rapid, labored respiration. The use of the remedy is confined to the first stage.

Spigelia is a remedy of the first importance in rheumatic and so-called idiopathic forms. It should be employed only in the earlier stages until effusion occurs, when it will be no longer indicated. Violent lancinating pain in the region of the heart is the most characteristic symptom pointing to its administration. Associated with this phenomenon there will be dyspnœa, palpitation, anxiety of countenance and irregular pulse. The remedy is recommended by some observers to be employed first.

Colchicum, in the form of the tincture or the alkaloid, colchicine 2x trituration ($\frac{1}{100}$ gr.), is not only to be recommended as a prophylactic of pericarditis during the course of acute rheumatic arthritis as mentioned, but also for the disease itself. It should be given in doses short of producing physiological effects. Its use is largely empirical. Pericarditis in association with rheumatism which has a preference for small joints points to its administration, while the presence of gout is a positive indication.

Bryonia is of service in the first and second stage of rheumatic or idiopathic pericarditis, especially when the exudation is plastic. It is suited to cases attended with high fever, pain of a sticking character, aggravated by the slightest movement, and occipital and frontal headache. It does not act well when the effusion is copious, nor when the pulse is feeble, irregular or intermittent. Its use is confined to the forms of pericardial inflammation above mentioned, as it is of no value in other varieties.

Asclepias tuberosa, according to Hale, will prove useful in some cases; its action is similar to that of bryonia. The special symptoms calling for its administration are mild fever, a dry, spasmodic cough and pain of a pricking character shooting to the left nipple and shoulder, accompanied with a sense of constriction around the heart. It is indicated in cases of a subacute type with effusion.

Asclepias syriaca is indicated when the disease is associated with nephritis with copious effusion and uræmic symptoms.

Cantharis is a remedy of a limited sphere of action in pericarditis. It is suited especially to types of inflammation which are marked by intensity of action and tendency to purulency.

Apis will prove of service in controlling the effusion of fluid and promoting its absorption. It is only therefore indicated in the later stages. Its action on the kidneys will also call for its administration in cases arising from renal disease.

Arsenicum will often prove effective when the stage of effusion is well developed, especially if it is of a serous character. It may be administered, however, irrespective of the character of the effusion. Hughes recommends it before the appearance of the exudation for the relief of the oppression and anxiety. Its special indications are violent and irregular palpitation, constrictive pain near the upper part of the sternum, with great anxiety and tendency to fainting, violent, tumultuous heart action, alternating with feeble and irregular action, and coldness of the extremities.

Potassium salts are useful in two forms, viz.: kali carb. in trituration, which may be used when the pain is of a sticking character and continues late in the disease, and potassium iodidum, which has proved effective in doses of five grains three times a day in promoting the absorption of fluid.

Cactus grandiflorus relieves congestion and irritability of the heart without causing depression. Its well-known characteristic symptom, the sense of constriction, as if the heart were grasped, in association with the phenomena of circulatory and respiratory disturbances peculiar to pericardial inflammation, is an indication for its use.

Sodii salicylas and *acidum salicylicum* are of utility in pericardial inflammation depending upon rheumatism. Beyond the presence of rheumatic infection their administration is entirely empirical.

Digitalis is of service during the stage of effusion, when the heart is feeble, with symptoms of cyanosis, dyspnœa and dropsy. In advanced cases, when symptoms point to impending failure of the heart action, five to ten drops of the tincture repeated at such intervals as the condition indicates will be called for. When speedy action is desired it should be administered hypodermically.

Morphiæ sulphas should be used when pain is very severe, when other means of relief have failed. It is best given hypodermically in small doses. Carefully administered it will not only relieve the pain, but will regulate the pulse.

When the effusion is excessive and the symptoms due to its presence urgent, paracentesis pericardii should be performed. The point of operating should be in the fourth or fifth interspace near the margin of the sternum. The tissue should be carefully divided with the scalpel until the pericardium comes in view; this should be punctured with a fine trocar or canula. Asepsis should be observed in all details.

Hæmorrhagic Pericarditis.

In a considerable number of cases of pericarditis the effusion contains more or less blood. This condition must not be confused with hæmopericardium, which is entirely different, denoting hæmorrhage into the pericardial sac independent of pericardial inflammation. Hæmorrhagic pericarditis, on the contrary, is simply a variety of an inflammatory process of the pericardium, characterized by sanguineous effusion. It is liable to occur in the course of cardiac and renal diseases, and especially in scorbutus and purpura, and in connection with chronic alcoholism, old age, depraved states of the system, tuberculosis and cancerous growths.

The clinical features of hæmorrhagic forms of pericarditis do not present anything markedly characteristic, and the symptoms and physical signs do not differ materially from those of non-hæmorrhagic types.

Purulent Pericarditis.

Ætiology.—Purulent pericarditis may occur as a result of change in the morbid process of sero-fibrinous inflammation or the effusion from the first may be of a distinctive purulent character. A large proportion of cases are associated with septico-pyæmia. Many again arise in connection with the acute infections. Tuberculosis may likewise prove a cause. The young are more subject to it than adults. According to their type the clinical features will vary.

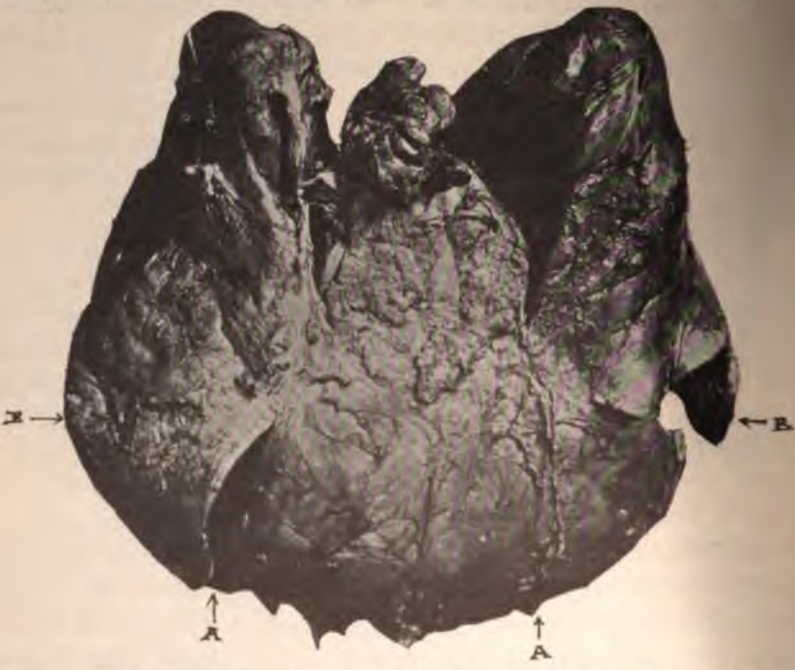

FIG. 1.—Pericarditis with purulent effusion secondary to empyema. The pericardium was greatly distended, as shown in the illustration, and contained 54 ounces of fluid. The empyema had been operated upon. The space between A and A represents the pericardium, B, the lungs.

Morbid Anatomy.—The first changes are similar to those of the sero-fibrinous variety. The pericardium presents a gray, granular surface, and the underlying myocardium is softened, fragile and pale. The exudation, needless to say, is purulent instead of serous.

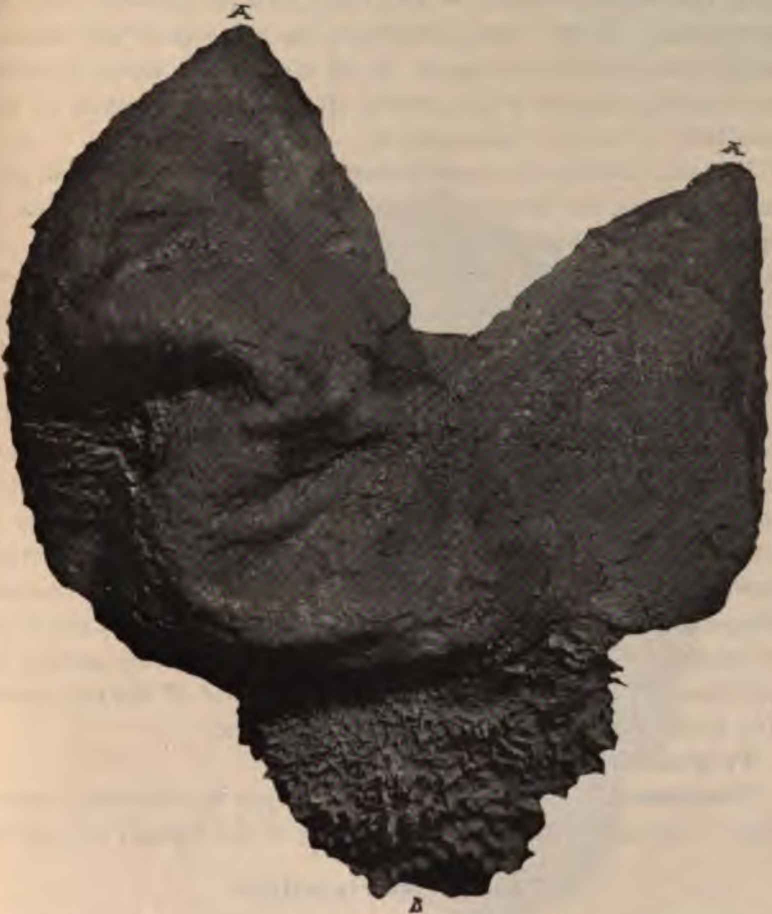

FIG. 2.—The same specimen as represented in Fig. 1. Here the distended pericardium, A–A, is opened and lifted up, showing the cor villosum.

Symptoms.—In those cases where the exudation has been primarily of a serous character, it is not difficult to recognize the appearance of purulent infection. In association with symptoms of pericardial inflammation there will be rigors and chills, followed by fever and excessive perspiration, hectic and emaciation. When, on the other hand, pericarditis is purulent in character from the onset, as in pyæmia and other septic diseases, it may be so disguised by the affection which it accompanies, or it may have so little effect upon the clinical features of the latter, that its presence may be overlooked. Under these conditions its recognition will depend solely upon the physical signs. In all cases of this nature a feeble, accelerated, irregular pulse should always direct attention to the possibility of cardiac complication.

PHYSICAL SIGNS.—In most instances the area of dulness on percussion will be less than in the case of sero-fibrinous pericarditis, as the purulent effusion is usually not so excessive. Otherwise, in both pericarditis with primary purulent infection and in the sero-fibrinous form which subsequently becomes purulent, the physical signs do not present anything especially distinctive from the ordinary type of the disease.

Diagnosis.—The presence of the physical signs of pericardial inflammation, with the occurrence of rigors, excessive perspiration and the temperature-curve denoting the presence of pus, will often enable a diagnosis to be made, especially when the ætiology is taken into consideration. It must be borne in mind, on the other hand, that purulent pericarditis may exist with an almost normal temperature, or even one subnormal, with little change in the pulse or respiration. Aspiration with a hypodermic needle should be performed to definitely determine the character of the exudation. The procedure is harmless when properly done.

Prognosis.—This is unfavorable.

Treatment.—The prompt removal of pus by incision is necessary. The treatment, therefore, belongs to the domain of surgery.

Chronic Pericarditis.

Ætiology.—Chronic pericarditis may arise as the consequence of acute inflammation, in which resolution has been arrested, or it may occur primarily as subacute or chronic. The distinction between the acute and chronic forms is not always clearly defined, for the condition may result from an acute inflammation of the pericardium which has existed only a brief period, as well as from one which has run a course of several weeks. In the first instance acute inflammatory diseases, such as rheumatism, pleurisy or mediastinitis, are the most probable precursors of the condition, while in the latter it gradually arises as a subacute inflammation in connection with Bright's disease, the exanthemata, tuberculosis, scorbutus, malnutrition and old age, and ultimately runs into the chronic form. The clinical features of those cases which occur as the result of acute pericarditis will be mainly those of that period of the stage of resolution at which the acute morbid process was arrested. When

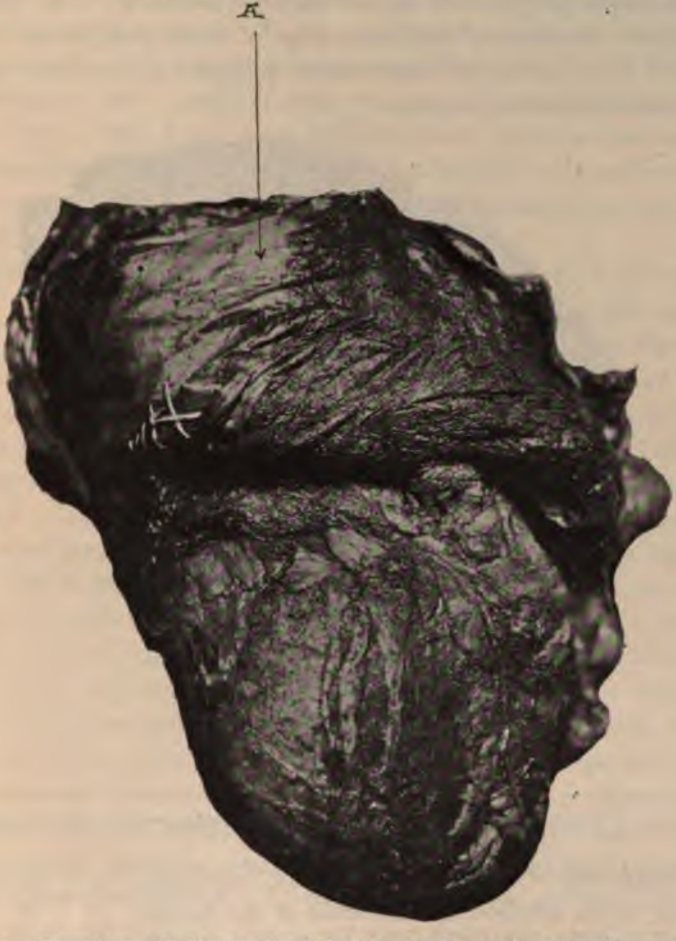

FIG. 3.—Chronic sero-fibrinous pericarditis with adhesions. A, pericardium detached and lifted up over base of the heart.

the disease from the beginning assumes a chronic form it is generally latent in its course.

Symptoms.—Symptoms are slow in developing and for the most part are those of the acute form, only less pronounced. The temperature will be normal or slightly elevated. The physical signs will be those of the latter stage of acute pericarditis characterized by weakness of heart action, increased area of dulness and occasionally friction murmurs.

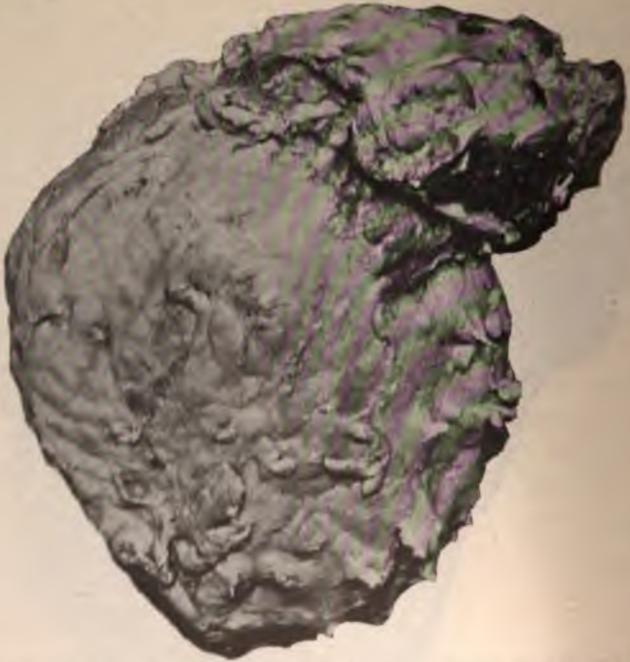

FIG. 4.—Chronic sero-fibrinous pericarditis showing roughened visceral pericardium.

Prognosis.—The prognosis is fair, depending upon the cause, nature of the effusion and duration of the disease.

Treatment.—Remedies for the removal of the fluid will be indicated as in the acute forms. When an excessive amount of effusion causes urgent symptoms paracentesis should be employed when other means have failed. The adhesions which may remain will not be influenced by treatment.

ADHERENT PERICARDIUM.

SYNONYM.—*Adhesive Pericarditis, Synechia Pericardii.*

Adherent pericardium is the existence of adhesions between the visceral and parietal layers of the pericardial membrane, arising from the presence of connective tissue formations.

Ætiology.—The condition may occur as the sequela of any variety of pericarditis, but is more liable to result from the fibrinous and sero-purulent forms, especially in connection with nephritis. Feeble heart action is conducive to formation of the adhesions, while increased energy tends to break them up.

Morbid Anatomy.—The morbid changes consist of fibrous bands of connective tissues which cause agglutination of the two pericardial surfaces with thickening of the pericardial membrane. Both these changes will vary in extent according to the nature of the primary inflammation. The degree of adhesion will vary from a few fibrous bands to complete obliteration of the sac, and the thickening of the membrane from that of a thin layer to one-quarter of an inch. The adhesions will take the form of bands or mats, and occur with greater frequency at the base of the heart than elsewhere. The newly formed tissue may undergo fatty degeneration or calcareous infiltration. The effect of adhesive bands upon the heart will obviously depend upon their extent and character. When they are loose and long, and there are no valvular lesions which may tend to produce hypertrophy, there will be little effect upon the organ. If, on the other hand, they are firm and short, especially if there are pleuro-pericardial adhesions, there will be a tendency to hypertrophy. When pericardial adhesions are associated with valvular disease, the heart is always enlarged and dilated with proneness to degenerative change. In cases of extensive and dense exudation calcareous deposits may occur after absorption, and form a hard case or shell partly or wholly enveloping the heart. Under these conditions the organ will be found atrophied with evidences of myocardial degeneration.

Symptoms.—The symptoms of pericardial adhesions are not distinctive. The lesion may exist without appreciable evidences, being revealed only by autopsy. On the other hand, it may give rise to marked and serious disorders of the heart. In mild vari ties functional disturbances are frequent, but as valvular disease often associated, the symptoms may be referable to the latter. I more severe cases, there will be dyspnœa, cyanosis and veno stasis; the pulse rapid, low in tension, and irregular. The puls paradoxus is sometimes present, but alone it is not diagnosti Bronchial and gastro-intestinal catarrh is frequent, and ascit

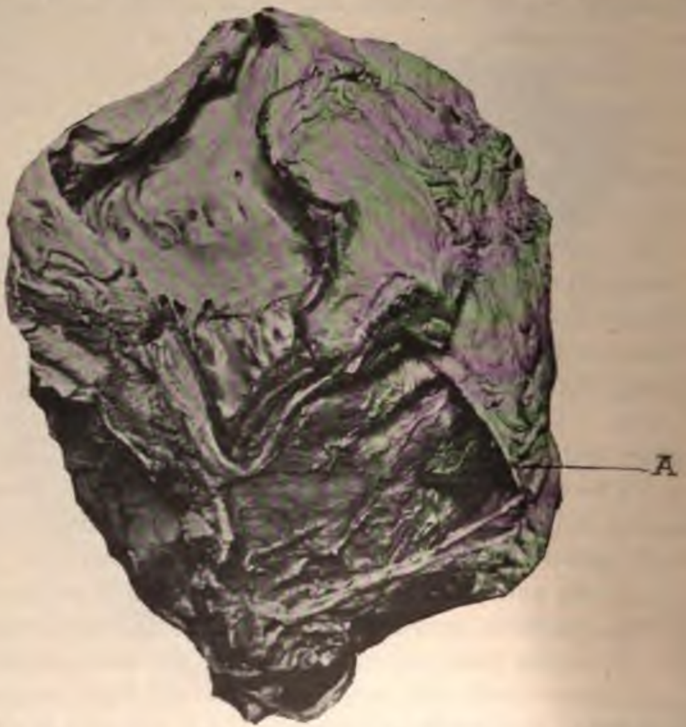

FIG. 5.—Adherent pericardium; A, a portion of the pericardium cut away and elevated. The heart was hypertrophied and weighed 46 ounces.

which has been attributed to the presence of bands pressing on the vena cava may arise. Occasionally symptoms of angina may also occur.

PHYSICAL SIGNS.—*Inspection* reveals important signs. The præcordium may show marked fulness, sometimes even to the deg of asymmetry owing to excessive enlargement of the heart. So writers speak of a flattening of the chest walls to the right and lef of the sternum. The extent of the area of the cardiac impulse is enlarged, and in some instances may seem to extend from the third to the sixth interspace. In extreme cases it may be observed from the right parasternal line to beyond the left nipple. The impulse is undulatory. An important sign is retraction with each beat of the heart, of the left half of the epigastrium, of the intercostal spaces surrounding the apex-beat, and of the lateral and posterior parts of the chest. This sign is unaffected by change of position, and is seen to better advantage in thin persons. In some cases retraction will appear marked at only one given point. Some describe the retraction as occurring at the apex-beat itself.

When the heart is adherent over a large area of the diaphragm, with each pulsation there is a systolic tug which may be transmitted through the diaphragm to its points of attachment. A systolic tugging therefore may be recognized in the region of the seventh or eighth rib in the left parasternal line. This may also be seen posteriorly on the left side over the eleventh and twelfth rib. On inspiration loss of normal expansion of the lower left side of the chest will be noticed. The inspiratory movements of the central portion of the abdomen will also be diminished, being equal to that of the lateral portion. In health the former is about two or three times that of the latter. On account of the attachment of the heart to the central tendon of the diaphragm, this portion may not descend with inspiration, and there is in consequence less visible movement in the epigastrium. The retraction of the lower portion of the sternum during inspiration is also sometimes observed. Another sign is collapse of the cervical veins during diastole, as pointed out by Friedriech, but this is of doubtful value. *Palpation* will enable the diastolic rebound of the heart to be more fully appreciated. This is regarded as a characteristic sign. There will be weakness of the apex-beat unless there is hypertrophy. Fixture of the apex-beat and immobility of the heart is sometimes observed.

Percussion will as a rule show an increased area of cardiac dulness dependent upon hypertrophy and dilatation which is present in the majority of instances. The increase of dulness will be lateral and is especially noticeable when there are valvular lesions associated with obliteration of the pericardial sac. A characteristic of the dulness is that it is unaffected by change of position, inspiration or expiration. *Auscultation* will not reveal distinctive signs. The heart-sounds are sometimes enfeebled, but when there is hypertrophy they will be intensified. Some writers mention a metallic character of the heart-sounds.

Diagnosis.—The recognition of the existence of pericardial adhesion and obliteration of the sac is difficult. The diagnosis will rest upon a group of symptoms and physical signs which must be taken collectively, rather than upon any one special feature. Immobility of the heart in varying positions and the diastolic rebound against the chest walls are valuable suggestive diagnostic points. Retraction of the epigastrium and intercostal spaces around the apex-beat and the diastolic collapse of the cervical veins occur in other conditions, but if these symptoms are found associated with the pulsus paradoxus the condition may be diagnosed as one of adherent pericardium. It is important also to note that adherent pericardium may exist without any of these signs.

Prognosis.—Adherent pericardium will present varying possibilities as to its future. Unaccompanied by complications, in itself it will permit of the functions of the heart being performed without serious interference, but if associated with valvular disease, as it generally is, grave pathological changes follow. In the latter condition the additional strain of more or less obliteration of the pericardial sac gives rise to a greater tendency to hypertrophy and dilatation, and consequent degenerative changes. Under these conditions sudden death from extreme weakness of the heart or interference with its functions is not unusual.

Treatment.—When the condition is a sequela of pericardial inflammation during the stage of resolution, remedial measures may be employed with a view of attempting to prevent the formation of adhesions. For this purpose apis, arsenic, potassium iodide and blister are recommended. When the heart is weak digitalis should be given. When adhesions have formed, remedies are without effect, and treatment [illegible] symptomatic. In mild cases of adhesion there [illegible] disturbance of the heart and all that will be called for [illegible] hygienic measures, careful diet and avoidance of [illegible] exertion. When the condition is more serious [illegible] indications pointing to weakness and failing action of [illegible] should comprise absolute rest, diet such as will not cause gastric irritation or distention, and remedies which act directly upon the heart in strengthening and increasing its action.

HYDROPERICARDIUM.

Hydropericardium is a serous exudation into the pericardial sac, occurring independently of inflammation. It is analogous to hydrothorax and ascites. A certain amount of serum is normal and the quantity found after death may bear little relation to that present during life, for if death is lingering the amount of fluid is augmented. To be pathological it must be more than two ounces.

Ætiology.—It is always a secondary affection arising in connection with general dropsy from any cause, but more particularly with cardiac and renal disease, and notably in chronic diseases of the liver. It also occurs as a sequela in the exanthemata and affections of the thoracic viscera which impede the circulation of the blood through the heart and lungs. In rare instances it may occur in scarlet fever, with few if any other symptoms of dropsy. Cachectic conditions may occasionally give rise to pericardial dropsy.

Morbid Anatomy.—Post-mortem examination will disclose a more or less distended pericardium. The serous surfaces will be free from evidences of inflammation, smooth, glittering and pale on account of the pressure on the vessels of the membrane. The pericardium may be thinned, especially if the fluid has accumulated gradually, or it may be thickened. The subserous tissues are pale, softened and œdematous, while the subepicardial adipose tissues will have disappeared to a great degree. The muscular tissue of the heart will be found pale and flabby; the right ventricle and right auricle will often be much dilated. Compression of the lower portion of the left lung and œdema of the cellular tissue of the mediastinum not infrequently occur.

Symptoms.—As hydropericardium arises as a result of other morbid changes, its symptoms and course are to a greater or less degree affected by those of the primary disease. In many instances its appearance is the forerunner of more complex pathological processes. Its symptoms arise from the pressure of the effusion, and are mainly characterized by dyspnœa and præcordial distress. The patient's position will be that frequently observed in cases of advanced cardiac disease. He will lean forward with the arms resting upon the knees, or seek to have the head held by an attendant. The pulse will be feeble, irregular and easily compressible. The veins of the neck will be prominent and turgid. If the effusion takes place gradually the disturbance will be less marked. Nervous symptoms are frequent and somnolence and muttering delirium may occur.

PHYSICAL SIGNS.—The signs will be those of effusion. There will be increased fulness in the præcordial region, with widening of the intercostal spaces; dulness on percussion which may assume the form of inflammatory pericardial effusion. The heart impulse will be feeble or no longer felt, even when the body is bent forward. The heart-sounds, including any adventitious murmur, are diminished in intensity. There will be a total absence of friction murmurs. Œdema at the base of the lungs, hydrothorax, ascites and enlargement of the liver are not infrequently observed as associated conditions.

Diagnosis.—The recognition of hydropericardium will depend upon the signs of effusion and the etiology in conjunction with the attending [illegible] symptoms and absence of a friction murmur. Hydropericardium must be distinguished from pericarditis with effusion by the absence of any history of pericarditis.

Prognosis.—This is decidedly unfavorable, except in cases of [illegible]

Treatment.—[illegible] directed chiefly to the original [illegible] practically but a symptom [illegible] The diet should [illegible] purgatives will [illegible] paracentesis of the peri-

HÆMOPERICARDIUM.

[illegible] effusion of [illegible] of pericar- [illegible] and clin- [illegible] matic, from [illegible] which may [illegible] ture of a [illegible] may occur when these vessels are in a state of profound degeneration, or rupture of the heart during some violent exertion, made possible primarily by structural change in the parenchyma of that organ. Among the traumatic causes may be mentioned wounds inflicted by sharp instruments, bullets, crushing accidents, or blows and falls. In the latter it is probable that disease of the heart walls is a predisposing cause.

Morbid Anatomy.—The condition will present markedly different features from those attending hæmorrhagic pericarditis. Post-mortem examination will reveal the source of blood and possibly changes in the pericardial membrane from disease of adjacent structures. The amount of blood will vary. In cases characterized by rapid development and immediate death, it will, as a rule, be small; in those where the hæmorrhage has been more gradual, and dissolution lingering, the quantity is larger. The effused blood may be either fluid or coagulated, but usually will be coagulated with some fluid blood remaining.

Symptoms.—The symptoms are those which arise from shock and compression of the heart by the effusion, and depend upon the rapidity with which the hæmorrhage occurs. There are two general types of cases: those which develop rapidly and terminate in sudden death, and those in which the course is more protracted and the patient lingers for a few days. In the former a person in apparent health, possibly engaged in some occupation, is suddenly stricken with unconsciousness, falls collapsed and pulseless and dies in a few moments, or at the most in a few hours. In those where the symptoms are not so rapid, the patient may complain of a sudden pain in the region of the heart, with a sensation of something having given way. Giddiness or faintness will accompany these symptoms, or they may be the first manifestations. The faintness will pass into unconsciousness and the giddiness may precede convulsions. The pulse will be feeble, irregular or absent, and the skin pallid, cold and bathed with perspiration.

Physical Signs.—Examination of the chest will reveal nothing characteristic. In those cases where the hæmorrhage has been gradual, physical examination may reveal increased area of dulness.

Diagnosis.—Hæmopericardium may be said to be characterized chiefly by the sudden appearance of collapse associated with the signs of increasing præcordial dulness, but a definite diagnosis cannot be made with any degree of certainty. When the symptoms have been preceded by the recognition of diseased conditions which are liable to give rise to such a lesion, it may be presumed that hæmorrhage into the pericardial sac has occurred.

Prognosis.—With few exceptions hæmopericardium is fatal.

Treatment.—There is little that can be done. In those cases which linger, stimulants should be administered, and the patient kept absolutely quiet.

PNEUMOPERICARDIUM.

Pneumopericardium is the presence of air or gas in the pericardial sac. Its occurrence is so rare that many clinicians of wide experience have never had an opportunity of observing a case.

Ætiology.—The causes are: (1) traumatism; (2) fistulous connection between the pericardial sac and adjacent air containing structures, as the result of disease, notably tuberculosis, cancer, empyema, abscess of the liver or mediastinum; (3) decomposition of purulent effusion. The latter is regarded by some observers as questionable. The bacillus aërogenes capsulatus, described by Nuttall and Flexner as the cause of subcutaneous emphysema in connection with wounds, is claimed to be capable of giving rise to the presence of gas in the serous cavities, hence it is regarded as a possible element in the causation of pneumopericardium.

Morbid Anatomy.—Unless the result of traumatism, pneumopericardium is almost always associated with pericardial inflammation, usually purulent, rarely hæmorrhagic. The pathological changes, therefore, will be those of pericarditis with purulent or hæmorrhagic exudation, in addition to the morbid processes which are the initial factors in its ætiology. The pericardial sac will usually be distended and bulged forward. On incision gas will escape with a hissing noise. In some instances the opening into the sac is free and in consequence there will be no distention. On laying open the pericardium, pus of a brownish, reddish or yellow color and fœtid odor will be found in varying quantities.

Symptoms.—The presence of pneumopericardium is chiefly characterized by pain in the region of the heart, sometimes of an agonizing nature, palpitation, dyspnœa, cyanosis, feeble, irregular and compressible pulse, and syncope. In some instances the appearance of the condition is marked by sudden collapse, which terminates in a few hours in death. In others the fatal result may be postponed for two or three days.

PHYSICAL SIGNS.—*Inspection* may show distention of the præcordial region, but this sign is not usual. *Palpation* shows feebleness or absence of impulse, which is a prominent symptom. *Percussion* reveals a striking feature, namely, areas of dulness and tympany which change with the position of the patient. When in the recumbent posture the tympanic percussion-sound will be elicited over almost the entire præcordium, while in an erect or sitting position the lower portion becomes dull. The area of tympany in some instances may also present variations. It may be coextensive with the præcordium or confined to the lower portion of the sternum and adjacent cartilages, while the upper portion gives the absolute dulness. *Auscultation* will reveal distinctive signs. The characteristic phenomenon is a metallic, splashing sound, heard with the pulsations of the heart, the action of which will be weak and irregular. These sounds are produced by the movements of the heart in the fluid and gas which fill the pericardial space and have been compared to that of a water-wheel. Friction murmurs are also frequently heard and endocardial murmurs may be present, but these are quite secondary to the splashing sounds which predominate over all. It is said the latter has been heard a distance from the body.

Diagnosis.—The tympanic percussion-note over the upper part of the præcordium or over its entire area, varying with the position of the patient, together with the splashing sound on auscultation, are characteristic signs which should exclude error. In connection with these striking features there will be the history of the disease, which, as a rule, will enable the diagnosis to be made without difficulty.

Dilatation of the stomach may sometimes be confused with pneumopericardium, from the fact that it will reveal an area of tympany on percussion, especially if forcible, and is sometimes accompanied by splashing heart-sounds. The differentiation can be made by noting that in dilatation of the stomach the upper part of the præcordium is never tympanitic, and that when the patient assumes a sitting position the splashing sounds disappear. Pneumothorax and pulmonary cavities on the left side are also mentioned as conditions possible to mistake for pneumopericardium, but the physical signs are too definite to admit of such an error.

Prognosis.—Cases arising from disease may die suddenly from syncope, or linger from a few hours to two or three days. When traumatism is the cause there are chances of recovery. If the wound is not septic, union may take place quickly and the air may be rapidly absorbed.

Treatment.—When the result of diseased processes, treatment should be palliative, with appropriate general and local antiseptic measures. In traumatic cases the strictest antisepsis should be adopted as early as possible in connection with remedies for sustaining the heart.

TUBERCULOSIS OF THE PERICARDIUM.

Tuberculosis of the pericardium or tuberculous pericarditis is uncommon. It may arise from extension of tuberculous disease of adjacent structures or it may form part of a general infection. As a primary disease its existence is denied, though some recent writers claim the contrary in cases of exceptional rarity.

Ætiology.—It may occur at any period of life from infancy to old age, but is more common between fifteen and thirty. Extension of tuberculous disease of the lungs, pleuræ, mediastinal and bronchial glands are the most frequent causes; the latter is a common source of infection. Many cases regarded as primary are due to unrecognized tuberculosis of these glands.

Morbid Anatomy.—The tubercular deposits are mostly found beneath the serous layers of the pericardium, either cardiac or parietal, and occasionally in the adhesions. They bear a strong resemblance to tuberculous disease elsewhere. The appearance of the pericardium will present two types of condition. In one there will be an exudation of fibrin and effusion, which is sometimes hæmorrhagic. The fibrin will present various degrees of organization and in the newly formed tissues there will be deposits of tubercular nodules. Microscopical examination will disclose the presence of giant cells in the organizing tissue. In a recorded case in which the pericardial inflammation had its origin in tuberculosis of the mediastinal glands, there was a purulent exudation within the sac containing tubercule bacilli, but no tubercular lesions of the pericardium itself were discovered, thus seeming to show that a secondary tuberculous pericarditis may arise without the presence of tubercles. In the other type of cases there are two forms, the miliary, which appears along the small vessels ramifying in the epicardial tissues and seen most distinctly in the sulci, and the larger masses of deposit, which are found more particularly about the base of the heart. These masses are gray externally, but yellow and cheesy on section. Ulcerations of the pericardial or epicardial structures occasionally may be observed, generally of a superficial character, but cases are on record where the ulcerations were deep, causing perforation and rupture of the heart muscle.

Symptoms.—Tuberculous pericarditis may be latent and unobserved, or it may give rise to decided phenomena, as in pericarditis of other types. The local symptoms and physical signs are those of the ordinary forms of the disease, in addition to the general symptoms of tuberculosis, such as excessive fluctuation of temperature, profuse perspiration, emaciation and weakness. A distinguishing feature is a tendency towards chronicity and persistence of symptoms.

Diagnosis.—The presence of the physical signs and symptoms of pericarditis in a tuberculous subject will point strongly, if not conclusively, to a tuberculous inflammation of the pericardial membrane, especially if the common factors in the ætiology of the non-tuberculous forms of the disease, such as rheumatism, Bright's disease or pleuro-pneumonia, are absent. In doubtful cases it is advisable to aspirate in order to establish the presence or absence of bacilli in the exudation; in fact, in many instances a positive diagnosis can only be established in this way.

Prognosis.—This is unfavorable.

Treatment.—This should be on general principles, employing every effort to sustain the strength and to combat the tuberculous tendency. Cardiac remedies will be especially indicated when there is a tendency to failure of the heart action. If effusion is excessive to the extent of seriously interfering with the functions of the heart, aspiration will be necessary.

SYPHILIS OF THE PERICARDIUM.

The pericardium is the least liable of all the heart structures to be attacked by syphilis. There are only a few cases on record which were of the late secondary and tertiary periods. Gummata are very rare. Mraček records only one case. More frequently the process consists of infiltration of the fibrous tissue with resulting adhesion. It is commonly associated with syphilitic affections of the myocardium, from which it becomes involved by extension.

There are no special symptoms by which syphilis of the pericardium can be determined, but if physical signs of pericarditis are present in association with evidences of syphilis, the condition may be regarded as warranting the suspicion that the pericardial lesion is syphilitic.

NEOPLASMS (CANCER, SARCOMA) OF THE PERICARDIUM.

Cancer and sarcoma of the pericardium are exceedingly rare. They are always secondary, arising either in connection with malignant disease in adjacent structures or following its development in distant organs.

Morbid Anatomy.—Growths from the heart may encroach upon the pericardial sac and involve its structure, but usually the pericardium becomes implicated from malignant disease of the mediastinal glands, œsophagus, pleuræ or lungs. New formations affecting the pericardium present the characteristic morbid changes of the disease which is present, accompanied by diffuse infiltration of the serous and subserous tissues. In exceptional cases cancerous nodules appear on the pericardium associated with similar conditions in other serous membranes secondary to cancerous disease elsewhere. In all conditions of morbid growths affecting the pericardium, an inflammatory process is liable to be induced—generally of a chronic and occasionally of an acute character.

Symptoms.—New formations of the pericardium are frequently unrecognizable during life, owing to the absence of symptoms or physical signs. On the other hand, there may be evidences of pericardial inflammation in connection with the general appearance of cachexia.

Diagnosis.—The diagnosis of these lesions will mainly depend upon the recognition of the diseased conditions in some other part of the body, in conjunction with interference with the functions of the trachea, œsophagus, and lungs.

Prognosis. The prognosis is decidedly unfavorable.

Treatment.—Treatment will obviously avail nothing. To meet the symptoms and palliate the consequences of the disease is all that can be done.

PARASITES OF THE PERICARDIUM.

In rare instances parasites may invade the pericardial membrane. Hydatid cysts have been found on post-mortem examination, but none have been recognized during life.

Actinomycosis affecting the pericardium is another pathological curiosity ; cases of this nature have been described, the implication of the pericardium being associated with the presence of the parasite in other parts.

Another rarity is the presence of free bodies in the pericardial sac. These were first described by Bouchard about thirty years ago. They were the result of detachment from a fringed-like substance which was found in the sac, somewhat like that found in the synovial membrane.

The diagnosis of these conditions is most always impossible. The recognition of the presence of the parasite in other organs in association with pericarditis should lead to the suspicion of implication of the pericardium.

CONGENITAL ANOMALIES OF THE PERICARDIUM.

Congenital defects of the pericardium are occasionally observed. The nature and rarity of the malformations do not permit of any classification. Defects generally only involve a part of the sac. Sometimes the heart lies with the lungs within the pleural sac, the visceral layer of the pericardial membrane enveloping the organ ; sometimes the pericardium has elongated openings through which the heart protrudes into the pleural cavity. Again, the pericardium is found to be generally adherent. Other anomalies which have been recorded are adhesions of the pericardium to the diaphragm, and pneumothorax with a congenital opening into the pericardium.

In some instances the pericardium may be absent. Diverticula have been reported ; they are, as a rule, developed from pressure of fluid within. They are formed of the serous layers of the membrane which protrude through a rupture of the fibrous layer. In some instances the diverticulum contains fluid.

Anomalies of the pericardium are not recognizable during life.

SECTION III.

DISEASES OF THE ENDOCARDIUM.

ENDOCARDITIS.

Endocarditis, or inflammation of the endocardium, is generally confined to the valves, though the mural layer may also be affected. The disease is classified as acute and chronic. The acute is further divided into two forms, simple acute or benign, and infective or malignant. The dividing line, however, between the benign and malignant varieties is by no means always clearly defined, the same micro-organisms occur in both, and not infrequently on post-mortem examination cases are found to be identical, which clinically had been considered to be of different types. For these reasons some writers disregard this division. Nevertheless, there are types which are distinctly benign, and which form the greater proportion of cases, and those which are distinctly malignant.

Simple Acute Endocarditis.

Synonyms.—*Benign, Papillary, Verrucose, Exudative and Rheumatic Endocarditis.*

Ætiology.—Simple acute endocarditis is a secondary affection; cases regarded as idiopathic in most instances are dependent upon some undetermined primal source. The disease may occur at any period of life, though it is generally confined to the young, principally for the reason that the affections with which it is associated are more prone to occur in youth and early adult life. A previous attack also predisposes to another.

Investigations all tend to support the claim that simple endocarditis shares with the malignant variety an infectious origin, or at least is attended with micro-organisms. Pyogenic organisms are frequently found on the diseased valves, especially the streptococcus and staphylococcus, though other cocci and bacilli are often observed. The streptococcus pyogenes is, according to Fränkel and Saenger, the chief excitant, although this view is not universally accepted. The pneumococcus, gonococcus, bacillus diphtheriæ, bacillus coli communis, bacillus anthracis, are also found. Certain special forms have been described, namely, the diphococcus endocarditidis encapsulatus and the micrococcus endocarditidis regatus of Weichelbaum, the bacillus endocarditidis griseus of Weichelbaum and Netter, and the bacillus immobilis et fœtidus of Fränkel and Saenger. In some instances only one variety is found; in others, several. When embolism occurs the secondary deposits will be attended with the same variety of organisms as are observed in the heart.

Latest researches in bacteriology compel the acceptance of the complete identity of the micro-organisms of both mild and malignant forms of endocarditis, yet it remains to be demonstrated why in one the destructive process is limited, while in the other it is so extensive. This difference may be explained as due to the greater virulence and abundance of the micro-organisms in the malignant variety.

As to the relation of the micro-organisms to those diseases commonly known to give rise to endocardial inflammation, we may, with our present knowledge of the subject, regard these bodies as specific poisons associated with the primary affection. It is quite probable that the micro-organisms, assisted by the friction produced by the current of the blood over the surface of the valves, fasten themselves upon the latter and give rise to the characteristic lesion.

Of the diseases which are recognized as the causes of endocarditis, acute articular rheumatism is the most frequent. It is variously estimated that from 40 to 80 per cent. of all cases are of rheumatic origin, and that fully one-third of the cases of acute rheumatism are complicated by endocarditis. It is obvious, therefore, that a close watch must be kept upon the heart in all acute rheumatic conditions. The occurrence of endocardial complications seems to be enhanced when many joints are affected, and when the rheumatic fever is intense, though this is not necessarily so, for they may arise during the course of the mildest forms of the disease.

When endocarditis occurs with acute articular rheumatism it usually appears in the first or second week of the disease. It may, on the other hand, be the first manifestation of the rheumatic process, the arthritic symptoms appearing later. Under these conditions the endocardial inflammation may be of pronounced intensity, while that of the joints is mild. Doubtless some of the so-called idiopathic cases are of this nature, the joint symptoms being so slight that they are almost, if not entirely, overlooked. Before leaving the subject of rheumatism as an ætiological factor in acute endocardial inflammation, its association with tonsillitis and with chorea must be borne in mind, thus showing that it may arise in connection with rheumatism in any of its forms, regardless of its severity or mildness, or whether it is typical or irregular in its manifestations.

Pneumonia is next in importance in the causation of endocarditis. It is not, however, a frequent cause. The large percentage due to rheumatism leaves but a small proportion to other causes. It is among these that pneumonia stands first. Bright's disease, especially chronic parenchymatous nephritis, must also be regarded as a prominent factor.

Other important causes are the specific fevers, notably scarlet fever, more rarely typhoid, diphtheria, measles, variola, varicella, and erysipelas.

Among other causes are chronic diseases attended with debility and anæmia, gout, chronic urethritis (gonorrhœal rheumatism), erythema nodosum, peliosis rheumatica, dyscrasia, diabetes, syphilis, extension of inflammation from the myo- or pericardium and local injury. Pregnancy and the puerperal state are also credited with exciting a recrudescence of acute inflammation in chronic cases. Tuberculosis must also be included in the ætiology of endocardial inflammation. Three varieties have been described: the granular, which is the most frequent, the caseous and the ulcerative.

Morbid Anatomy. The disease is characterized chiefly by the formation of vegetation on the segment of the valves, varying in size from the most minute bodies to that of a pea. These excrescences are found on the surface of the valves opposed to the blood current about two or three millimetres from the margin. They have been compared to a row of beads. The first textural change which takes place is reddening of the serous membrane. This is followed by opacity and thickening, especially on the free surface of the valves, causing them to present an elastic feeling and swollen appearance. This change is due to infiltration of the endocardium with young cells, the process beginning in the layer of flat cells. The new formative cells are developed from those of the layer beneath the endocardium, and from the leucocytes. This hyperplasia is attended with softening of the deeper layers of the intercellular structure and as the process is continued the intercellular substance is also involved. The endothelial cells likewise play an important part by assuming a tendency to proliferate. They push forward the endocardium and form papillary vegetations. These elevations are surrounded in the deeper layers of the endocardium by proliferations of tissue which is not distinctly limited, but which shows a tendency to hyperplastic formation from the periphery to the centre. There is no exudation on the villous projections, but coagula are found upon them, for, acting like foreign bodies, the fibrin from the blood which is separable during the process of acute inflammation becomes deposited upon them. In this way the villi may assume considerable size, some conical in shape and some round. At first they are very small and numerous, so as to present a granulated appearance, but later they may become as large as peas. Micro-organisms are often found in these fibrinous deposits. The new formations obviously will interfere with the passage of the blood through the valvular orifices.

Another feature of endocardial inflammation is that it is almost always confined to the left side of the heart, except in fœtal life, when the contrary is the case. This is generally attributed in part to the fact that the freshly oxygenated blood is more favorable to the micro-organisms concerned in the inflammatory process, and that after birth the blood in the left side of the heart is more completely oxygenated, while in fœtal life this is the case in the right side. Moreover, the left side of the heart is more active and subjected to greater pressure, while in the fœtal life the right side is subjected to these influences. The inflammatory process is further characterized by a tendency to secondary sclerotic changes, which causes contraction, shortening and deformity of the valves and valvular openings, as described under chronic endocarditis.

The seat of lesion corresponds to the point of maximum contact but the mitral valve is much more frequently involved than the aortic. In some instances the chordæ tendineæ are also implicated and in exceptional cases the inflammatory changes will be confined to these structures alone. Endocardial inflammation often involves the muscular structure of the heart. This myocarditis may extend to some depth and by weakening the heart walls give rise to acute dilatation. In some instances it may cause ventricular aneurysm.

During the course of acute endocardial inflammation there is always a possibility of the formation of emboli. This is more likely to occur when the vegetations are large as they are then more liable to become detached by the force of the blood and carried to various parts of the body, particularly the brain, spleen and kidneys, where they give rise to embolic infarcts. This serious complication is more frequently observed when acute endocarditis is superimposed upon a chronic inflammation. The embolic processes are as a rule benign and simply cause obstruction in contradistinction to those associated with malignant endocarditis when they form metastatic abscesses.

Symptoms.—The clinical history of simple acute endocarditis is distinguished in many instances by the uncertainty and negative nature of its symptoms in contrast with the serious results which frequently follow its occurrence. The disease will often exist without giving evidence of subjective symptoms. In many instances the only evidence of its presence is the development of a murmur. Taking into consideration the insidious nature of the onset of endocardial inflammation, it is obviously a matter of the greatest importance that a close surveillance should be kept upon the heart during the course of any disease which is recognized as a possible factor in its causation. Pre-eminently is this the case during the acute articular rheumatism. While there is often absence of subjective phenomena, which could be regarded as distinctive, certain symptoms may appear; these are pain, dyspnœa, augmentation of fever, and disturbance of the heart action. Such symptoms in the course of a disease liable to excite endocarditis are presumptive evidences of the presence of the affection.

Pain is not a marked symptom. It is more a feature of peri- than endocardial inflammation. When present it is generally of a dull, aching character. In some cases it is referred to the epigastrium, especially in children in whom the tendency to pericardial complications is more marked.

Distress in the region of the heart, in the form of a sense of constriction or weight, is sometimes observed, or the patient may complain of being constantly aware of the heart action from the strong pulsations which it imparts. The pulse at first is strong and full; later it becomes feeble and irregular. As the disease progresses the strength of the heart action will be diminished, as evidenced by the more feeble character of the pulse; at the same time the heart itself will appear active, thus showing that while its irritability increases, its propulsive force is weakened.

Dyspnœa is a variable symptom. There is usually increase of respiration, but true dyspnœa is only observed, as a rule, in severe forms, where it may be pronounced with symptoms indicating deficient aëration and feeble circulation.

In some instances the rheumatic process may expend itself principally and almost exclusively on the endocardium, the articular manifestation being so slight as to almost escape notice. This is illustrated in the case described in connection with Figs. 6, 7 and 8.

The temperature may rise abruptly during the course of the primary disease. It is seldom, however, more than 102° or 103° F., but may rise higher.

In the rare instances when emboli follow serious complications may occur according to the location of the embolus. This condition, however, is much more frequent in the malignant form of the disease. An important feature of simple endocarditis is the possibility of its assuming the malignant type. This, however, does not often occur. The duration of the disease is variable. It may run its course in two or three weeks, or continue for several months.

PHYSICAL SIGNS.—*Inspection* may show at the commencement of the disease increase of the area of visible impulse and irregularity of the heart action. Later these signs may be indistinct. The patient will usually lie on his back or left side. *Palpation*, in addition to confirming the signs detectable by inspection, shows the heart acts with greater frequency and force, but subsequently if the walls are weakened by acute dilatation it will be diminished in force. *Percussion* will not disclose any change unless the cavities of the

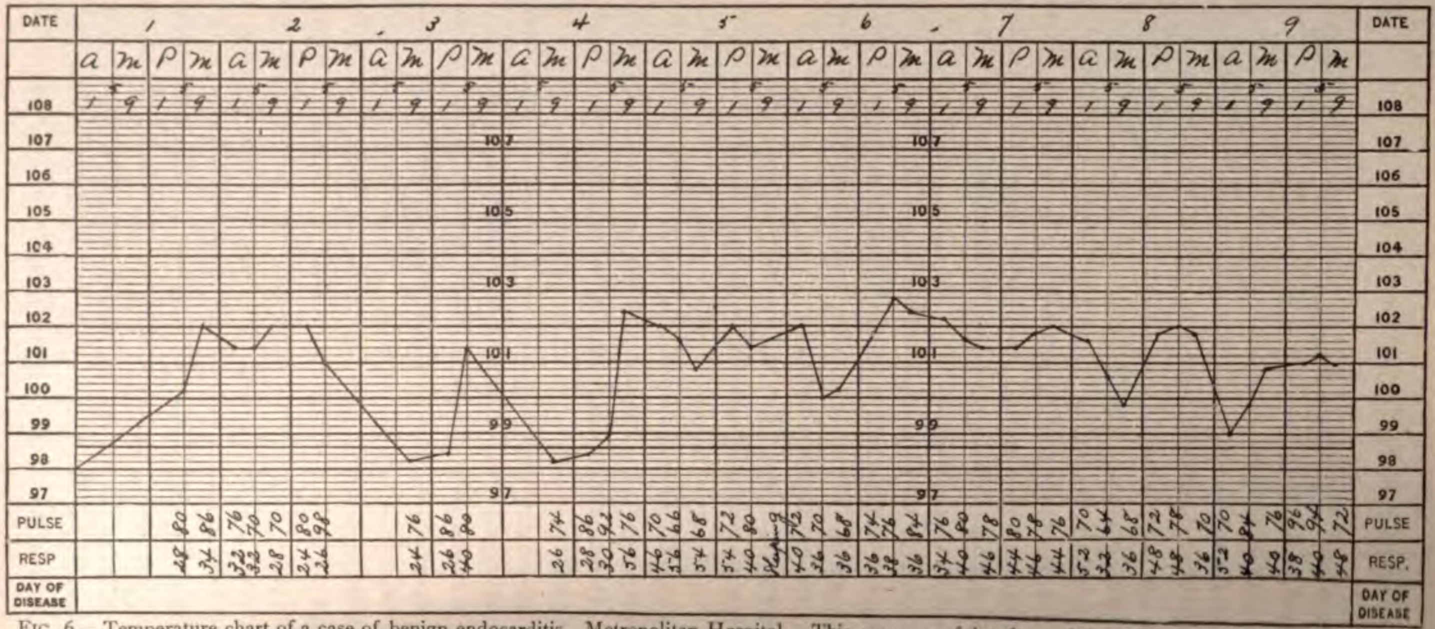

FIG. 6.—Temperature chart of a case of benign endocarditis. Metropolitan Hospital. This case was of that form of the disease in which the rheumatic infection primarily and almost exclusively manifested itself upon the endocardium. The only articular involvement was that of the little finger of the left hand, which was so slight as to almost escape attention. In addition to the temperature, there was dyspnœa, slight præcordial pain, cyanosis and marked double aortic and mitral systolic murmurs. The aortic insufficiency predominated, being accompanied by the characteristic water-hammer pulse. There was the history of a former illness of a similar nature showing that the endocarditis was superimposed upon a previous lesion. The patient was a male, aged 22 years. Fig. 15 represents the pulse-tracings of this case.

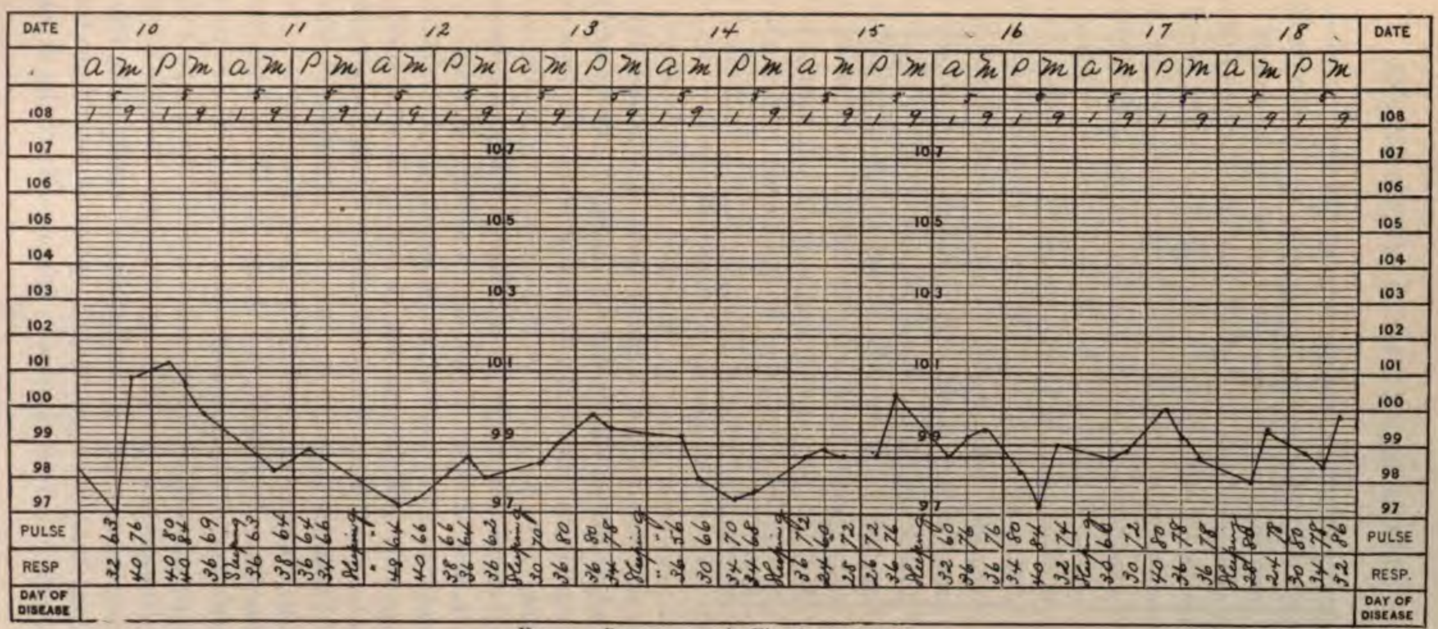

FIG. 7.—Same case as in Fig. 6, continued.

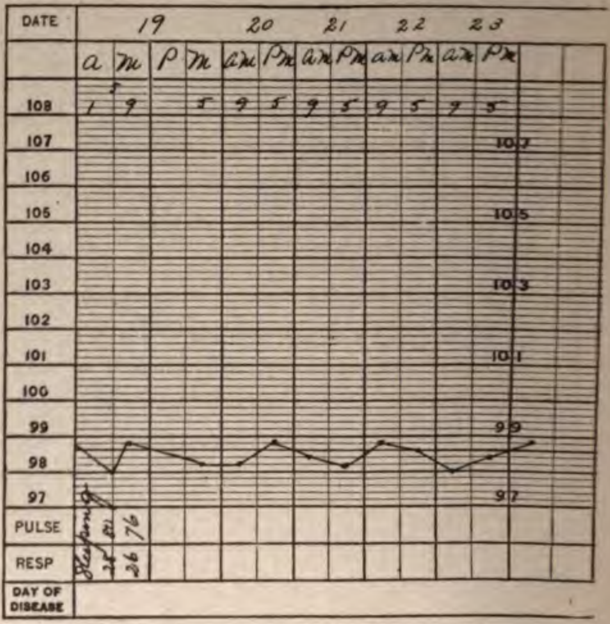

FIG. 8.—Same case as in Fig. 6, concluded.

heart become distended with blood from feebleness of the cardiac muscle, when there will be slight increase of præcordial dulness. This change when observed will be confined to the later stages of the disease.

Auscultation furnishes the most reliable evidence of the presence of endocarditis, although at the best it is often negative. A loud, soft-blowing, systolic murmur will be heard over the valve or orifice affected, usually with the greatest intensity at the apex, owing to the fact that in most instances the seat of lesion is at the mitral segments. When produced at the mitral or tricuspid orifice, in addition to the changes in the endocardium, there may be an insufficiency, for the reason that there is shortening of the chordæ tendineæ and tumefaction of the margins of the valves. If the aortic valve is the seat, roughening of the endocardium of the ventricle will be the cause. Under these conditions the murmur will be heard at the second interspace at the right edge of the sternum. Basic murmurs may also be heard in the area of the pulmonary orifice to the left of the sternum, but are functional in origin. Before the development of the murmur there will be prolongation of the first sound of the heart with consequent shortening between the first and second sound. This is a sign of incipient endocarditis. It arises from mitral insufficiency or from roughening of the mitral curtains. When due to the former there will be increase in intensity of the second sound at the pulmonary orifice. All diagnostic murmurs will grow in intensity as the disease advances. Diastolic murmurs occur, but are exceptional.

A very important characteristic of recent endocardial murmurs is the limited area of transmission. This is owing to the fact that in the earlier stages of the disease the inflammatory structural changes are not sufficiently organized or dense to permit of the transmission of murmurs. The presence of cardiac murmurs, even when associated with symptoms of acute disease, is not always positive evidence of acute endocarditis, for it has been demonstrated that degenerative changes in the muscular structures of the heart may lead to enfeeblement of the walls and papillary muscles, thus causing imperfect action of the valves. For the same reason the muscular tissue of the orifices may undergo changes which alter the size of the openings and prevent perfect closure of the valvular segments.

X-RAY DIAGNOSIS, see Part VII.

Diagnosis.—The diagnosis will rest on the physical signs, though these are not absolutely invariably trustworthy, but when taken in connection with the symptoms usually associated with endocarditis, they constitute a more reliable basis of diagnosis. A positive indication of acute endocarditis is the development of a murmur which is found, after repeated examination, to have developed during the course of a disease recognized as an element in the ætiology of endocardial inflammation. A soft, bellows-like murmur may be heard in acute febrile disease, yet an autopsy will show freedom from endocardial implication. Murmurs of this nature are functional, and are mostly heard over the aortic area, while those of endocarditis are generally heard over the mitral area.

Simple acute endocarditis can be distinguished from pericarditis by the difference in the physical signs. In endocarditis the murmur is more diffuse and is not intensified by bending forward or by pressure of the stethoscope, and is not so superficial as in pericarditis. Again, the signs of effusion with diminution of the cardiac impulse and absence of the friction murmur are phenomena present in pericarditis, but absent in endocarditis. The cöexistence of pericarditis and endocarditis must be borne in mind. Old endocardial murmurs may be confused with recent, but the latter are more generally characterized by the limited area of diffusion and absence of hypertrophy.

Aortitis, though rare, may lead to confusion in the diagnosis of endocarditis. It is characterized by a more rapid pulse and the prominence of pain, which appears along the spine and præcordial region, and is aggravated by motion.

Prognosis.—The outlook is favorable as regards life, as most patients recover from simple acute endocarditis, but the inflammatory process frequently results in deformities of the valves and orifices, leaving the heart in a permanently impaired condition. Permanent lesions are less liable to occur in children than in adults, but in early childhood the outlook is more serious on account of the frequency of pericardial complications, which may prove fatal, and the greater liability of the recurrence of rheumatism, which may further damage the heart structures. The fact that the affection may assume a malignant type should be borne in mind. The possibility of embolism of the different viscera as manifested by hemiplegia, enlargement of the spleen and albuminuria, complications which frequently terminate unfavorable cases, must also be taken into consideration.

Treatment.—Inasmuch as endocarditis in the great majority of instances appears secondarily in connection with other affections, the first question of treatment is the efficacy of prophylaxis. The frequency of the implication of the serous membranes of the heart in acute articular rheumatism, especially the endocardium, renders the question of prevention during the course of that affection one of paramount importance, especially when the serious sequelæ are taken into consideration. The preventive measures directed against complications of this nature, as discussed under the treatment of pericardial inflammation, applies with equal force to endocarditis. Of these measures, the internal administration of colchicine short of physiological action and the application of cold to the præcordium commend themselves to the writer. Caton, of Edinburgh,* strongly advocates the application of a succession of small blister

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* On the Prevention of Valvular Disease, Richard Caton, M.D., Edinburgh Medi Journal, April, 1899.

The salicylates, while of undoubted value in acute articular rheumatism, are often disappointing as preventives of endocarditis. The alkaline treatment is endorsed by the older writers; one to three drachms of the bicarbonate of potassium in aqueous solution may be administered daily until the urine becomes alkaline.

The importance of rest cannot be too greatly emphasized. Patients should be kept in bed as quiet as possible, and every effort which might tax the heart should be avoided. The sick room should be free from draughts, but well ventilated. Flannels should be applied over the chest and blankets may be substituted for sheets. The diet may be liberal but light, consisting mainly of milk. Stimulants may be necessary and should be given freely if the heart shows indications of weakness. Hot applications to the præcordial region are useful adjuvants for the relief of pain. On the other hand, cold in the form of ice-bags or Leiter's coil may be preferred.

Warm baths at a temperature of 90° to 100° F., repeated from one to three times during the twenty-four hours, will prove of service for their sedative effect upon the heart and nervous-system.

All treatment is necessarily influenced by the primary disease. In the instance of acute articular rheumatism the salicylates are of undoubted utility.

The following remedies prescribed according to their especial indications should constitute the principle line of treatment.

Aconitum is useful in the first stage in sthenic cases, when there is high fever, anxiety, restlessness, pain and tendency to pulmonary congestion.

Veratrum viride may be used in the early stages in preference to aconite, when the heart action is more violent, with symptoms of pulmonary congestion, without the restlessness and anxiety which indicates aconite. It may be given when the pulse is quick or the reverse.

Ferrum phosphoricum may be given in place of aconite in the early stages when there is fever, soft, full, quick pulse, shooting pains, worse on motion, and absence of the irritability which usually calls for aconite. It appears to act better in the young.

Bryonia will prove of service, especially in rheumatic cases, when there is pain of a sticking character. It is regarded as effective in preventing the exudation of lymph. It may be advantageously administered in alternation with aconite, or in association with the salicylates, that is, during the intervals between the doses of the latter.

Belladonna is a remedy which will be called for when there are symptoms of irritation and congestion of the brain. It acts best when the first stage is passing into the second, and when there are symptoms of præcordial aching, throbbing beneath the sternum, with strong pulsations which jar the head and neck. The heart action will seem strong with irregular contraction. The remedy is often indicated in children.

Colchicum is a remedy of great importance in rheumatic and gouty cases, especially when aconite has failed. Violent action of the heart, with strong pulsations, excessive palpitation with tearing pain in the præcordial region, quick, small and thready pulse, point to its selection. The tincture or the alkaloid should be given. When using the latter, either as a preventive of endocarditis during acute articular rheumatism or for the attack itself, give three to five drops of a solution of one grain to one ounce of alcohol, every two or three hours, decreasing the dose if physiological effects are produced.*

Cactus grandiflorus is useful in nervous palpitations of the disease. Pain and oppression and the well known characteristic sensation of the heart being grasped are its chief indications.

Spigelia is of special value in cases of rheumatic origin with pain in the region of the apex, of a knife-like character, with tendency to radiation.

Kalmia is indicated by the presence of shooting, stabbing pains in the heart, with great dyspnœa and feeble pulse. Rheumatic endocarditis with pain shifting from one point to another.

Rhus tox is indicated in those forms of endocarditis which accompany articular rheumatism where the joint pains, notwithstanding their severity, induce restlessness and cause the patient to attempt to move about. A tendency to assume the typhoid or malignant form is another important indication.

Arsenicum is usually regarded as limited to endocarditis of Bright's disease and that of the malignant type. While it does not control inflammation, it will prove useful in the paroxysms of palpitation and attacks of cardiac syncope. It may also be given in the later periods when dyspnœa, weak, irregular pulse, mental agitation, anxiety, anasarca, hepatic congestion and albuminuria are prominent symptoms.

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* Practice of Medicine, W. C. Goodno, Philadelphia, 1897.

Digitalis must be given with caution, if at all. When the myocardium is involved its use is attended with danger.

Other remedies are cimicifuga, pulsatilla, caulophyllum, ledum and arnica.

The salts of ammonium, particularly the carbonate, may be administered continuously with a view of obviating intracardial coagulation of the blood. If this accident should occur, strychnine and alcoholic stimulants should be freely given in conjunction with the ammonium.

During convalescence complete rest should be maintained. The patient must be kept in bed until after all acute symptoms have subsided, and for some weeks after apparent recovery he should remain perfectly quiet, so as to avoid any strain upon the valves or heart walls. This is especially imperative when murmurs remain. During this period, such remedies as aurum, graphites, potassium iodide and sulphur may be administered with a view of causing absorption of the products of inflammation.

Malignant Endocarditis.

SYNONYMS.—*Acute Infectious Endocarditis; Ulcerative Endocarditis; Mycotic Endocarditis; Diphtheritic Endocarditis.*

Malignant endocarditis is an infectious disease of comparative rarity, characterized by suppuration and ulceration of the valves of the heart, and the presence of micro-organisms of virulent activity. It usually develops in the course of some primary infectious or septic disease amid symptoms of a severe type. In exceptional cases it may apparently occur idiopathically.

Ætiology.—The infectious nature of malignant endocarditis is very decided and has been recognized since Koch discovered the bacillus of tuberculosis in 1882. The diseases which enter into its causation are: Pneumonia, rheumatism, the specific infectious fevers, empyema, diphtheria, septicæmia, pyæmia, puerperal fever,

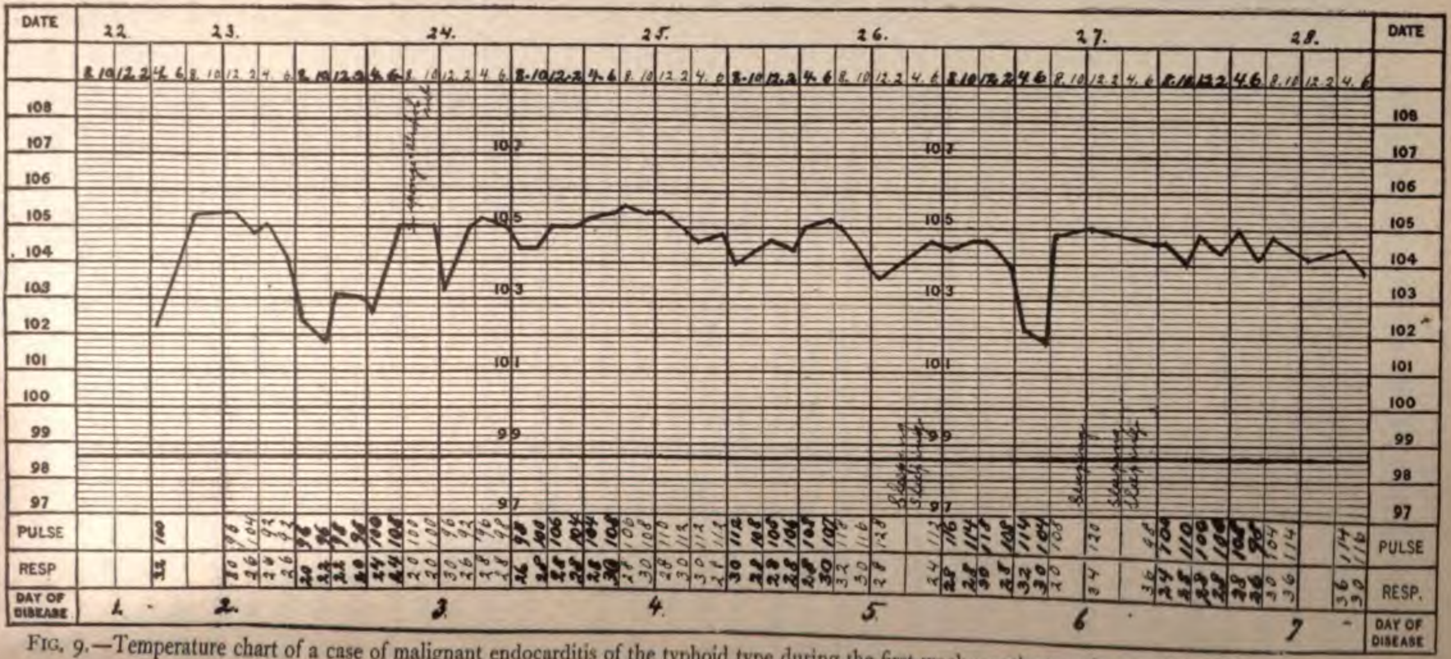

FIG. 9.—Temperature chart of a case of malignant endocarditis of the typhoid type during the first week; patient, male, aged 36. Metropolitan Hospital. The case terminated fatally at the end of the sixth week.

gonorrhœa, Bright's disease, dysentery and malarial infection. Thus it will be observed that the conditions which may give rise to malignant endocarditis resemble to a certain extent those which produce the milder form, but, as a rule, the former originates in connection with septic diseases, while the latter, in most instances, occurs as a complication of acute rheumatism and diseases of a less infectious character. The nature of the lesion, however, is not necessarily determined by the form of the original affection, for malignant types may be excited by rheumatism, and the mild may be caused by sepsis. Pneumonia is a more frequent cause than was formerly supposed, as endocarditis when associated with it is apt to be of the malignant type. In septic diseases the malignant endocardial process may be either simply a part of the general disease or a local manifestation. Sometimes it constitutes the chief pathological factor on account of the affinity of the micro-organisms for attacking the valves of the heart. The presence of chronic valvulitis is an important predisposing cause. In sixty-nine fatal cases Goodhart found this lesion present in sixty-one ; according to other observers it is less frequent.

Malignant endocarditis, as stated elsewhere, shares with the simple form the same bacteriology, but no satisfactory reason has as yet been assigned for its greater malignancy, nor for the change of the milder form into the malignant. The specific irritant is probably the streptococcus pyogenes, though the pneumococcus, staphylococcus, gonococcus, bacillus coli, bacillus anthracis and certain special forms as mentioned under the ætiology of the milder variety are also found.

Morbid Anatomy.—The pathological changes are generally located in the left side of the heart. The mitral valve on its auricular surface, and the aortic on its ventricular, are most frequently involved. In rare instances the pathological process is confined to the right side.

There are three types of lesion : the vegetative, suppurative and ulcerative. In the early stages vegetations, such as are observed in the milder forms of the disease, appear on the segments of the valves. These vary in size from a pin head to a pea, and are of a grayish-yellow color. They possess a strong tendency to necrosis, which is liable to spread and destroy more or less of the endocardium. Histologically these bodies are composed of granulation tissue covered with granular and fibrillated fibrin, containing numerous micro-organisms. Their interior is prone to suppurate and to the formation of abscesses, which rupture and result either in perforation or in aneurysmal sacs, the result of subsequent distention of the abscess cavities with blood. Ulcerations are also frequent; they may be only slight erosions, but often are deep and result in perforation. In some instances the walls of the heart become involved in the same pathological processes as are observed in the valves, or mural and valvular lesions may be combined. This condition is not common.

The ulcerative process may cause great destruction, and involve the chordæ tendineæ, and perforate the septum or even the ventricle. A prominent feature is the formation of emboli. The vegetations become loosened, and are carried by the circulation to different parts of the body, giving rise to infarctions and emboli, which are especially liable to locate in the spleen and kidneys, and with less frequency in the cerebral meninges, intestines and integument. Sometimes these detached bodies will be of sufficient size to obstruct large vessels. Saturated with the products of inflammation, they form suppurating infarcts wherever they locate. They vary in number from two or three to several hundred. In some cases they will cause softening of the coats of the vessels at the point where they become arrested, and give rise to aneurysmal dilatations. The serous cavities and joints will also become involved, and examination will show the presence of purulent exudation.

Symptoms.—Diversity of clinical manifestations and absence of distinctive features in the serious phenomena which accompany the disease is characteristic of malignant endocarditis. Errors in diagnosis are not infrequent; in many instances the lesion has remained unrecognized until revealed by the autopsy. The fact that the disease arises secondarily and often forms a component part of a general infection frequently obscures the true condition. At the same time symptoms indicating the presence of a grave lesion are always present, although those which point distinctly to the heart may be slight or entirely absent; as, for example, when the disease occurs as a complication of pneumonia, meningitis or empyema. Cases of this type are those which are likely to remain unrecognized during life.

In others heart symptoms are more pronounced. There may be præcordial pain, palpitation, oppression and disturbances of the circulation. These symptoms are more likely to be observed when the condition arises in connection with chronic valvular disease and rheumatism.

Intensity of the inflammatory process, general infection, emboli and unfavorable prognosis are features which distinguish malignant endocarditis from the milder form of the disease. The frequency of embolic complications which may involve any organ is especially characteristic.

From the standpoint of symptomatology the disease is described under four forms, namely: the septic, the typhoid, the cerebral and the cardiac. It is also classified as acute, subacute and chronic. The acute follows the septic, typhoid and cerebral varieties; the subacute and chronic, the cardiac.

The septic form, as the term implies, is that which arises from septic and pyæmic diseases, and is more commonly observed in connection with puerperal fever, and infected wounds and inflammations. It presents the general symptoms of septicæmia and septico-pyæmia, being ushered in with severe rigors and chills, followed by a temperature which runs high, but which fluctuates to a marked degree. As the temperature falls there is profuse perspiration. These symptoms, commonly observed in all forms of sepsis, are usually pronounced in this variety of malignant endocarditis. The fact that they are recurrent frequently suggests simulation of malarial poisoning. The pulse will be extremely variable and will undergo rapid change, ranging all the way from one hundred to one hundred and fifty per minute. During the pyrexia it will be compressible and often dicrotic and irregular, while during the intervals of the febrile attack it may fall to normal or subnormal. Digestive disturbances are pronounced, there will be no desire for food, coated tongue, diarrhœa, and tympanitic distention. General prostration and anæmia will be rapid. Examination of the heart may reveal the presence of murmurs, or all evidence of any valvular defects may be absent. Systolic murmurs in the mitral and tricuspid regions are common and valuable aids to diagnosis, although not, as a rule, at all characteristic, as the same phenomena may be heard during any disease attended with high fever.

The heart impulse in most all cases will appear feeble and the area of dulness will show general enlargement. There may be dyspnœa, palpitation and cyanosis, especially if there are mural changes.

The typhoid form bears a strong resemblance to its prototype and might easily be mistaken for it, were it not that its onset is different, being attended with severe chills and rigors, which appear suddenly in the course of the original disease with which the endocardial complication is associated. The temperature in these cases will be high and will continue so with uneven fluctuations. The pulse at first will be regular, but later is quick, compressible and dicrotic, the respirations quick and superficial. An eruption similar to that of typhoid may appear, but is not identical with it. Sordes collects on the teeth, the tongue may be red with furred edges in the earlier stages, or it may be cracked and fissured; later it is the dry, brown tongue usually seen in typhoid states. In rare instances the parotid and salivary glands will swell. Prostration will be rapid and nervous symptoms, such as delirium, stupor, and coma will follow. As the disease advances, diarrhœa and meteorism will appear. Pain in the region of the heart may be present, and sometimes there will be oppression and cyanosis, but, as a rule, cardiac symptoms are few.

The cerebral form includes those cases in which the brain and meninges are involved. The symptoms resemble those of meningitis and cerebro-spinal meningitis. Frequently it is difficult to realize that cerebral and meningeal lesions are not the sole causes. In cases of this type associated with pneumonia, pneumococci have been found in the meningeal exudation.

The cardiac type includes a large number of cases. They are those which have previously been subject to chronic valvular disease, and are characterized by a tendency to run a subacute or chronic course which may continue for several months. The onset is insidious. The patient complains of malaise, is weak and anæmic. There will be fever with exacerbations, which may be the first symptom noticed. In other cases the affection may appear like rheumatic arthritis. After these symptoms have continued for a few days, rigors, followed by fever and profuse perspiration, will appear. The temperature may rise to 104° F. and fall shortly to normal. There may be an interval during which the temperature remains normal, then the rigors and fever will return. Sometimes this train of symptoms continues for weeks and even months. A resemblance to malaria will also be noticed in this form. Some cases will undergo distinctive valvular changes and thus remove any doubt as to the nature of the affection.

The presence of emboli, which has previously been mentioned as a prominent feature of malignant endocarditis, is common to all varieties. Hence, in conjunction with each type of the disease as described, the possible occurrence of symptoms which characterize the presence of these phenomena in various parts of the organism must always be taken into consideration.

Sudden hemiplegia points to cerebral embolism; pain in the lumbar region with hæmaturia indicates the kidneys are the seat. Embolic inflammation occurs in the meninges, pleuræ, lungs, peritoneum and joints, and various affections of the mucous membrane arise from the same source. The skin is not only the seat of varieties of rashes, but sudden purpuric blotches also occur which are of grave significance and important aids to diagnosis. Other symptoms common to all varieties are enlargement of the spleen and albuminuria. Abscess of the parotid gland and retinal hæmorrhages occur in rare instances.

Physical Signs.—These may be entirely negative, but in the majority of instances a systolic murmur will be heard. Increase of the area of cardiac dulness is common. Indefiniteness, however, must be said to characterize the physical signs of malignant endocarditis. Murmurs are difficult to locate with any degree of accuracy, and when heard they may vary from day to day. The adventitious sounds which are most frequent are muffled, systolic, mitral and aortic murmurs, reduplication of the first sound of the heart, and soft systolic murmurs over the pulmonary and aortic valves.

The presence of systolic murmurs considered in conjunction with symptoms which point to the presence of malignant endocardial inflammation may be regarded as a valuable aid in diagnosis. The second sound of the heart will also sometimes be accentuated, even when no lesion has been recognized. Chronic valvular disease may be present with its characteristic signs and those of hypertrophy; also pericarditis with its friction murmur, which may appear as an additional complication.

Diagnosis.—It may be readily understood from the complex symptomatology and often vague and illy-defined physical signs that the recognition of malignant endocarditis is at times attended with difficulty. The previous history and the circumstances attending each case must always be carefully considered. Severe rigors, early high temperature and embolism, if accompanied by symptoms indicating acute endocardial complications, are sufficient to enable the diagnosis to be made readily. Again, when the history is clear, the presence of rigors and high fever, with symptoms of embolism, may be regarded as suggestive of malignant endocarditis, even in the absence of symptoms pointing to the heart. Emboli, it must be borne in mind, while a characteristic feature of malignant endocarditis, is also observed in the mild form of the disease, but to a much more limited degree. Moreover, in the malignant form, emboli give rise to metastatic abscesses, while in the simple or benign they only cause obstruction.

A continuous fever, fluctuating and appearing like that of malaria, with the presence of a cardiac murmur, or even in the absence of any abnormal heart-sound, may be regarded as suggestive of malignant endocarditis, provided there are no evidences of suppuration and the possibility of malarial infection has been eliminated by the proof of the absence of plasmodium malariæ in the blood.

In the cardiac variety the diagnosis is attended with less difficulty. The presence of rigors, high fever, with extreme fluctuations and profuse perspiration in cases which are the subjects of chronic valvular diseases, together with the history, will usually enable a diagnosis to be made in most cases.

The diseases with which malignant endocarditis are more likely to be confounded are, septicæmia and pyæmia, malaria, acute tuberculosis, meningitis and typhoid fever.

From septic and pyæmic diseases it is frequently impossible to make any differentiation, for the endocardial implication may constitute simply a part of the general infection. In all forms of septic disease, whether complicated with endocarditis or not, there is a characteristic temperature, pulse and skin. The only means of ascertaining the presence of endocarditis under these circumstances is the manifestation of cardiac symptoms and the physical signs of a cardiac lesion.

A bacteriological examination of the blood is the most positive means of diagnosing cases of septico-pyæmic origin. To make the examination, proceed as follows: Disinfect the finger of the patient first by washing with soap, ether and bichloride solution. Sterilize the lancet, then puncture the finger, and receive the blood on a platinum needle, which has been previously sterilized by heat and thoroughly cooled. Cultures may then be made as deemed most convenient. When larger quantities of blood are desirable, use a fine, hollow needle, such as is usually employed for intravenous injection. Draw the blood from a vein which has been rendered prominent by bandaging. An important point in these examinations is that the blood should be drawn during the chill or height of the fever, as the micro-organisms are most abundant at this period. The result of examination will always show the presence of pyogenic organisms in septico-pyæmia.

Intermittent quotidian and tertian malarial fevers, especially those varieties which arise in highly malarial climates and which follow a protracted course, may present many of the appearances of malignant endocardial inflammation of the cardiac type. In these instances malignant endocarditis may be distinguished by the absence of plasmodium malariæ in the blood, by the negative effect of quinine, and in some cases by the presence of embolism and the physical signs of cardiac disease.

Acute tuberculosis is not so liable to be confused with endocarditis, from the fact that there are often clinical features which will remove all elements of doubt. In acute tuberculosis there are frequently manifestations of the implication of certain organs, more especially the lungs and brain. In the former, examination will reveal the signs of consolidation and the presence of tubercle bacilli in the sputum. Again, in general tuberculosis the fluctuations of the pulse and temperature are more uniform, the perspiration shows a decided tendency to come on at night, and the emaciation is more rapid, while the absence of any pathological physical signs of cardiac lesion is a feature.

Purulent meningitis bears such a strong resemblance to the cerebral form of the disease that no differential diagnosis is possible, unless the history of the case or the presence of cardiac symptoms will enable a distinction to be made.

The typhoid form presents many points of similarity to typhoid fever; at the same time there are marked differences which may be summarized as follows:

MALIGNANT ENDOCARDITIS.	TYPHOID FEVER.
Some disease liable to cause endocardial complications present.	Condition of patient generally good before attack.. In some instances there is a history of an epidemic.
Rarely, if ever, primary.	Always primary.
No prodroma.	Prodroma marked.
Onset sudden in the course of a disease liable to cause it.	Onset usually insidious. No other disease, as a rule, present.
Commences with severe rigors and chills.	Chills, if present, are very slight.
Temperature rises with great rapidity, falls and rises again, with marked fluctuations.	Temperature rises slowly, fluctuations not so marked, progressive step-like increase.
Blood shows signs of leucocytes.	Blood shows decrease of leucocytes and the presence of typhoid bacillus.
Diazo reaction rare in urine in any disease but typhoid fever.	Diazo-reaction in about 90 per cent. of typhoid cases. An important element in diagnosis, especially when considered in conjunction with clinical history.
Widal test negative.	Widal test positive.
Embolism a feature of the disease.	Embolism rare.
Loud systolic murmurs often present.	Soft systolic murmurs may sometimes be observed.

Certain infective diseases, such as variola, cerebro-spinal meningitis, may sometimes present clinical phenomena so closely resembling those of malignant endocarditis, that it is only after a certain time has elapsed that the conditions can be differentiated.

Finally, there are cases in which the symptoms are so indefinite that the recognition of the true condition is impossible.

Prognosis.—This is decidedly unfavorable. Most cases run their course at the most in five or six weeks. Some terminate fatally in a few days. The subacute form may continue for several months and sometimes recover.

Treatment.—Treatment has not been attended with favorable results. Rest and quiet should be enjoined and the strength supported by a liberal and frequent administration of concentrated liquid foods and alcoholic stimulants. Strychnine, strophanthus and digitalis will be necessary to sustain the heart.

Lachesis is of service in severe forms of endocardial complications which occur in connection with septico-pyæmic diseases and malignant infectious fevers. It is indicated by great depression of the nerve-centres, especially of the pneumogastric, as evidenced by cardiac, laryngeal and bronchial irritation, ecchymoses, hæmorrhages from the mucous surfaces, disorganization of the blood, a constant sense of suffocation, dyspnœa, rapid, feeble, intermittent pulse and a general typhoid state.

Crotalus is recommended for the same types of the disease. It possesses indications very similar to those of lachesis, but with greater intensity of action. Profound depression of the nerve-centres, rapid disorganization of the fibrin of the blood, ecchymoses, mucous hæmorrhages, low muttering delirium and coma are symptoms which point to its administration.

Naja is mentioned by some writers, but does not appear to have been used to any considerable extent.

Arsenicum will at times prove of service in the typhoid conditions when there is great restlessness, gastro-enteric irritation, profound prostration and other symptoms which especially indicate administration of the drug.

Other important remedies are baptisia, bryonia, rhus, acidum muriaticum, belladonna, hyoscyamus and agaracine.

The antiseptic remedies, such as quinine, salol, resorcin, naphthol, the salicylates, and the sulpho-carbolates, have been very generally employed. Of these, quinine seems to have met with the most general approval. It should be given in full dose, five to twenty grains every four hours, during the stage of the decline of fever. Dr. Jousset, of Paris, recommends aconite in full doses of the tincture to be given during the stage of fever in conjunction with the quinine during the apyrexia.

Serum Therapy.—The treatment which bids fair to lend a brighter aspect to the prognosis of malignant endocarditis is serum-therapy. While it is true that the method is still to a certain extent in the stage of experimentation, there are sufficient evidences of its success to warrant its use in serious cases of septico-pyæmic origin, especially as at the present time it appears to be the most hopeful resource in these serious forms of disease.

Antistreptococci serum is antitoxin which has been used in grave forms of septic infection, including those associated with endocarditis. It is recommended by some writers to be given in all varieties of septicæmia, but according to Marmorek* the action of the serum is limited to those varieties of sepsis which arise from the presence of the streptococcus pyogenes, the most common form of septicæmia; infections due to the staphylococcus, the bacillus coli communis, the gonococcus, and the Talamon-Fraenkel coccus are not amenable to the action of the serum. The remedy is given by subcutaneous injection into the areolar tissue of the lateral abdominal wall. The syringe and skin should previously receive the most thorough sterilization and antisepsis, respectively. The surface should be prepared by washing with an ethereal solution of soap, then washed with bichloride, 1–1000, and sprinkled with boric acid. The dose ranges from 30 to 45 c.c. in twenty-four hours. The usual dose is first 15 c.c., and after twelve hours 10 c.c.; in severe cases 25 c.c. for the first dose, repeated at the end of twenty-four hours. The British Institute of Preventive Medicine recommends 20 c.c. for the initial dose, followed by the same quantity if the temperature does not fall. At present the practitioner must rely upon the instructions which accompany each article of serum.

Collargolum, which has been comparatively recently successfully employed, may be used in all forms of septicæmia. In endocarditis, when serious symptoms of septico-pyæmic infection predominate, it is a remedy which should be considered. It is a non-poisonous allotropic silver which is permanently soluble in water and albuminous fluids, and is an effective general antiseptic which can be introduced into the system without causing either local reaction or general poisonous effects. It remains metallic silver in sterile blood or lymph; but in the presence of pathogenic bacteria or their toxins it enters into combination and acts as an antitoxic agent. Introduced into the system by inunction of the ointment or hypodermically, or by the mouth in capsules, or in solution, or applied locally to wounds, it enters the lymphatics and circulates dissolved in the blood. The fæces frequently become brownish-black, but argyria never occurs. Collargolum is usually best employed in the form of the unguentum Credé as an inunction; any surface of the body may be selected, preferably the back, buttocks and loins. The single dose for adults is 3 grams (45 grains); for adolescents, 2 grams (30 grains); and for children, 1 gram (15 grains); thoroughly rubbed into the cleansed skin for from twenty to thirty minutes, and not washed off. Three inunctions upon three successive days are required in all cases, and the ointment is to be used subsequently as indicated. The remedy has been successfully employed in pure and mixed forms of infections of staphylococcus and streptococcus and other bacteria. In recent cases it is said to exert a rapid curative action, and in those of a more chronic nature it is also effective, provided it is used before secondary changes in the vital organs have occurred. It has been employed in conjunction with injections of antistreptococci serum.

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* Lancet, ii., 1896, p. 1076. Lancet, i., 1897, pp. 520, 1264. Lancet, ii., 1897, pp. 92, 907, 1339.

Normal saline solution in the form of intravenous or subcutaneous injections have given favorable results in infections of all varieties. As it is not likely that the small amount of saline fluid which is introduced into the circulation can produce destruction of the toxins, it is probable that a renewal of physiological action is induced. Whatever the action, this agent has proved to be a means of saving life.

During the administration of antistreptococci serum and collargolum remedies to maintain the heart should be administered. Of these, strychnine stands first; $\frac{1}{30}$ to $\frac{1}{40}$ grain should be given every two to four hours, according to indications.

CHRONIC VALVULAR DISEASE OF THE HEART.

SYNONYMS.—*Chronic Endocarditis, Chronic Interstitial Endocarditis, Chronic Valvulitis, Heart Disease.*

Valvular disease of the heart signifies all conditions of imperfect action of the valvular mechanism which arise from structural changes. These lesions include the morbid processes which destroy the integrity of the valves and orifices, those which involve the papillary muscles and chordæ tendineæ, and those which affect the heart walls and lead to dilatation and consequent incompetency. In the last instance the valves themselves may sometimes much less frequent occurrence. In fatal cases the evidences of endocarditis are found in inflammatory deposits on the valves. This certainly points to rheumatism as the common causal factor. A case recently observed by the writer showed the presence of a mitral systolic murmur and hypertrophy which followed chorea, which in turn developed after tonsillitis. There were no other manifestation of rheumatism. This case illustrates very aptly the presence of endocarditis in the more unusual forms of rheumatic infection, as well as its association with chorea.

Erythema nodosum, according to Trousseau, may occur with endocarditis, and there does not appear to be any doubt but that the former is a manifestation of rheumatic infection.

Chronic endocarditis and atheroma, after acute endocarditis, are the chief sources of valvular disease. These conditions occur for the most part during middle and after-life, and clinically are impossible to separate. They are chronic from their incipiency and are mainly dependent upon the presence in the blood of toxic and chemical irritants, namely, uric acid, alcohol, lead, syphilis, and upon the effects of mechanical causes. These groups of ætiological factors all tend to degenerative changes of an interstitial and atheromatous nature. In some instances such changes are simply part of the retrogressive influence of age, while in others they are the results of arthritic and renal disease. Persons of a gouty or rheumatic diathesis may develop chronic valvular disease without ever experiencing any manifestations of acute arthritic disturbance. Bright's disease may, in like manner, set up chronic interstitial changes. It also is a fact that Bright's disease may arise from valvular affections of the heart as well as be their cause. Alcohol may act by inducing changes which are the results of its irritant effects on the endocardial surfaces giving rise to chronic inflammation, or by constant over-stimulation it may produce dilatation, hypertrophy and interstitial degeneration, or it may give rise to diathetic conditions which are liable to excite endocardial changes.

Degenerative processes which originate in the heart walls, either from strain or from loss of elasticity of the heart muscle, are the result of atrophic and other retrogressive changes. These conditions may also affect the papillary muscles and chordæ tendineæ, causing stretching in some parts and shrinking in others. The latter is most frequent. The natural outcome is dilatation with inadequacy of the auriculo-ventricular valves, owing either to distortion of their attachments or widening of the orifices; the valves themselves under these circumstances may either remain intact or may participate in the degenerative process. Dilatation of the heart from any cause may prevent proper want of closure of the valves and consequent insufficiency. Atheroma plays an important part in these changes.

Mechanical influences include muscular strain, as induced by any long-continued muscular effort and direct violence, and are important causal factors in the production of degeneration of the heart structures. In the first instance dilatation and valvular insufficiency may occur, or chronic endocardial and atheromatous changes of the valves and myocardium may arise. The primary causes of the degenerative processes lie in the undue pressure to which the aorta is subjected by occupations which call for great exertion or which necessitate working in a constrained position, as in the case of miners, hammerers and porters, as well as among athletes who over-train, and those who suddenly endeavor to perform some feat requiring a great amount of prolonged muscular action. Strong muscular efforts which require holding the breath and fixation of the chest walls and prolonged closure of the epiglottis, necessarily cause compression of the contents of the thorax, and in consequence their effect is exerted on the heart. Under these circumstances the column of blood in the aorta is put under high pressure and a greater effort on the part of the valves will be required; at the same time the increase in pressure in the aorta will demand more force from the ventricles to expel their contents; hence more pressure is also put upon the mitral valves and chordæ tendineæ. The constant and continuous effect of these forces in time will give rise to degenerative changes.

High arterial tension as observed in renal disease, gout and other diathetic conditions will have a similar effect. The strain on the valve is not, it is true, so intense as under the conditions mentioned, but is more continuous and incessant; and as this state of the circulation is frequent in middle age it accounts for a large proportion of cases which occur at this time of life.

Injury to the valves is an occasional, but unusual, cause. It may much less frequent occurrence. In fatal cases the evidences of endocarditis are found in inflammatory deposits on the valves. This certainly points to rheumatism as the common causal factor. A case recently observed by the writer showed the presence of a mitral systolic murmur and hypertrophy which followed chorea which in turn developed after tonsillitis. There were no other manifestation of rheumatism. This case illustrates very aptly the presence of endocarditis in the more unusual forms of rheumatic infection, as well as its association with chorea.

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Injury to the valves is an occasional, but unusual, cause. It may arise from direct violence from some external agency, or rupture may occur spontaneously from muscular effort on the part of the patient, such as lifting, violent attempts of respiration and forcible coughing. The rupture may be complete or partial, and is more likely to involve the aortic valve.

Hæmorrhage of the valves is a possible, but rare, cause. The pulmonary valves, from the nature of their structure, cannot, in all probability, be affected, but the auriculo-ventricular valves from their vascularity may become the seat of a hæmorrhage which forms the starting-point of valvular disease. Clinically, this condition is obscure.

Morbid Anatomy.—The pathological processes operative in chronic diseases of the valves and orifices of the heart are thickening, sclerosis, adhesion and atheroma. The usual seat of lesion is the endocardium of the valves and orifices, but it may also be that of the heart walls, from whence the valve structures become implicated by extension. When the valves are primarily involved the changes will frequently be confined to the free margins or the bases of the segments. When seated on the auriculo-ventricular valves the inflammatory changes will first appear on the auricular surface; when on the semilunar valves they will be observed on the ventricular side.

Thickening is the first visible change. It is the immediate effect of an overgrowth of fibrous tissue which arises from inflammation, in which there is proliferation of the endothelial cells and a round cell infiltration of the subendothelial tissue, which becomes organized into fibrous tissue. This change first makes its appearance on the free borders of the leaflets. The next change is that of contraction and induration which follows as the result of the presence of hyperplasia. The shrinkage of the newly formed tissues causes shortening of the curtains of the valves and curls the margins of the cusps. Thus a perfect adaptation of the segments to the valvular openings will not be possible and insufficiency is the result. At the same time the deformed valves may cause obstruction. Under these conditions the aortic cusps are often rolled back and adherent, though their edges may remain movable. In the case of the mitral valve the tendinous attachment of the papillary muscle frequently contracts and draws the valve out of place.

The adhesive process first begins at the base of the segments and may extend upwards and lead to constriction. Excessive adhesion of the valvular segments along their free margins may reduce the orifice to a button-hole, as observed from the auricular surface. This condition may occur at the aortic orifice to a moderate degree, but is more common at the mitral orifice, except in the case of children.

Vegetations on the cusps and at the orifice are often the cause of obstruction. They will be found upon the surface most exposed to the flow of the blood-current. They vary in size and are attended with shrinkage and contraction.

Degenerative changes constitute an important element in the pathology of all forms of valvular disease. They may follow the chronic inflammatory process in the endocardium or may arise in the heart walls and secondarily impair the functions of the valves. In the case of the endocardium sclerosed patches are often associated with fatty and calcareous changes. The former are more particularly observed on the endocardium of the left side. They also appear on the opposed surfaces of the cusps and give rise to disturbances. Calcareous degeneration is observed in old cases, either in localized areas or coexistent with the diseased tissue. A lime-like infiltration of the adherent sclerosed cusps may occur which, in extreme cases, may convert the valves into a calcified mass. Bony rings are also found which further impede the mobility of the valves.

Fatty degeneration leading to necrotic ulceration and destruction of tissue is frequent in old cases, especially on the mitral and aortic cusps. This type is characterized by some writers as *endocarditis chronica malignans.*

Aneurysmal dilatations may also occur in the substance of the valves. They arise from the effect of blood-pressure upon ulcerations, and are more commonly observed associated with the mitral valve. Rupture of one of the chordæ tendineæ may occasionally be found as a result of ulceration or aneurysmal dilatation.

When the aortic valves are the seat of lesion, the changes brought about by the morbid processes, as above described, will give rise to an obstruction to the out-flowing blood-current, and at the same time, owing to the inability of the segments to close perfectly, a diastolic back flow into the left ventricle occurs. The aortic ring to which the valvular segments are attached will also become implicated, undergoing thickening, sclerosis and, finally, atheromatous degeneration. Similar changes will be observed in the aorta, where thickened atheromatous patches will generally be found in the ascending arch and also in the aortic segments. Extension of the sclerosis and atheroma to the coronary arteries constitute a very important feature.

Changes of the same nature occur when the mitral valves are involved, the deformities which are produced resulting in insufficiency and stenosis. As the diseased conditions advance they attack the papillary muscles and chordæ tendineæ, which undergo contraction and rigidity. This results in not only affecting the valves, but causes narrowing of the orifice. In the early period of mild forms of mitral stenosis a circle of vegetations will appear on the auricular surface of the mitral orifice, the edges of which subsequently become hardened. These changes will in time become involved in a sclerotic process which will make it impossible for the valves to be forced back against the ventricular walls during diastole on account of the cohesion of the valvular structures and the contraction of the chordæ tendineæ, which draw the leaflets of the valves toward the apex of the heart.

Implication of the endocardium of the walls and of the heart muscle itself arises much more frequently from extension of the inflammatory process from the valves than from primary involvement. The seat of lesion, under these circumstances, discloses grayish-white, elevated patches which involve the underlying muscular structure. This condition favors the deposition of fibrin, and the presence of an ulcerated surface or fibrinous deposit on the valves will account for the formation of emboli. The embolic processes, as in acute endocarditis, will generally appear in the spleen, brain, kidneys or liver. In cases of long standing, muscular degeneration will generally be found in the form of sclerosed patches and fibroid, fatty and calcareous changes. These types of myocardial degeneration will more frequently be observed in aortic areas, especially when there are atheromatous changes in the coronary arteries.

When the function of the valves suffer interference from causes apart from those connected directly with the cusps which may remain normal, the morbid changes operative are those which arise from structural changes in the myocardium, papillary muscles and chordæ tendineæ. The ventricles may be so dilated that there is want of correspondence between the valvular structures and the heart chambers, or the papillary muscles and chordæ tendineæ undergo atrophic degenerative processes which diminish their functions. Stretching of the valvular openings may also occur from severe strain, which produces loss of tone and elasticity of the heart muscle. These conditions result in auriculo-ventricular insufficiency, and are called relative incompetence.

In case of disease of the right side of the heart the morbid processes are of a similar nature. A separate discussion therefore is not necessary.

As a consequence of these morbid anatomical changes in the heart, a long series of phenomena manifest themselves. Some are intimately associated with the heart itself. and some are the more direct results of interference with the general circulation. All are of important significance.

Obstructive lesions are attended with diminution of the blood-supply beyond the point of interference of blood-flow. This will result in a lessening of the pressure and a lowering of nutrition in the obstructed area and consequent hypertrophy.

Insufficiency is characterized by a loss of blood-pressure beyond the affected locality in consequence of the regurgitation. The effect is more marked in the peripheral circulation.

Symptoms.—A valvular lesion may manifest itself by distinctive symptoms or it may remain latent, being discoverable only on post-mortem examination. The latter is more likely to occur when the disease arises in connection with atheroma or sclerotic endocarditis. These conditions may escape recognition from the fact that they are exceedingly subtile in their course, and, owing to a favorable condition of the patient, compensation is often readily established and maintained. When endocardial changes are limited to the walls and the valves remain intact, chronic inflammatory and degenerative changes may exist for a long period without giving rise to symptoms. Sometimes the presence of these conditions will only be made known by the products of inflammation being thrown off in the circulations and locating as emboli in the brain, lungs, kidneys, spleen or retina, where they are the sources of profound disturbances.

In many instances the origin of valvular disease is insidious and the morbid condition may persist for a considerable period without attracting attention. In a large number of cases the presence of a murmur will be the only symptom, and the patient will be unconscious of anything wrong with the heart. Many cases of this nature will continue unchanged for an indefinite period, while others owing either to the progress of the disease or to some extraneous influence, or to the combined effects of both, will develop symptoms, the future depending on the ability of compensation to maintain itself. It may therefore be said that there are two classes of cases in valvular disease : those in which compensation is adequate, which do not present any symptoms other than the physical signs, and those in which compensation shows evidences of weakening as manifested by the development of grave symptoms.

ONSET AND EARLY SYMPTOMS OF FAILING COMPENSATION.—Notwithstanding it is important to know the period when the disease process began to invade the valve structures, this knowledge will, in most instances, be limited to the cases which can be traced to the consequences of acute endocardial inflammation, notably those arising in connection with rheumatism. The early symptoms may appear in a variety of types and will present variations not only according to the extent of the damage to the valvular structures, but also according to the particular valve involved.

The most frequent type of early symptoms of valvular disease is when the patient seeks advice for relief from shortness of breath and palpitation which comes on without any apparent cause or which is excited by emotion or slight exertion. In addition, there may be pain and a sense of distress referable to the præcordial region.

Another type is when the patient complains either of a sense of fulness or constriction in the region of the heart or of vague anomalous sensations of disturbances which are referred to the præcordial region. It is very important to recall that while these symptoms may point to the presence of a cardiac lesion, they may also be present in connection with nervous and gastric disturbances in the absence of any organic change in the heart.

In some instances the attention will be excited by failure in the general health ; there will be loss of vigor and anæmia, and fatigue on exertion will be quickly experienced. Sometimes there will be aching of the legs, languor and a tendency to drowsiness. The mind will also undergo a change ; matters which were of interest will cease to be so and there will be a disposition to become indifferent, despondent, morose or irritable. In connection with this train of symptoms there will be dyspnœa and palpitation attending any unusual effort.

Œdema of the ankles and feet will be the first symptom observed in some cases. The patients will complain that their shoes have become uncomfortably tight or there is a little puffiness about the shoetops. Sometimes the swelling is observed only at night, disappearing entirely in the morning. There will probably be also in association with these symptoms some shortness of breath which, however, may not have been sufficiently marked to cause much disturbance.

In a certain number of cases advice will first be sought for gastric disturbances, headache and vertigo, which on examination will be found associated with organic change in the heart.

Another symptom which may first excite suspicion is the condition of the kidneys, more especially on account of the excess of urea with deposits of urates in the urine.

In others pulmonary symptoms will be the first to appear and point to the lungs rather than the heart as the seat of trouble. The presence of hæmoptysis, which is more liable to occur in conditions of high tension of the pulmonary circulation and in embolism of the lungs, especially in association with mitral stenosis, may prove especially misleading, but a careful examination of the sputum, together with the presence or absence of fever and the effect of digitalis, will usually enable a diagnosis to be made. It must be remembered, however, that tuberculosis and valvular disease may coëxist.

In some instances, especially in persons of advanced life, in whom a valvular lesion has existed for many years, grave symptoms of failing heart power may come on suddenly from slight exciting causes and sometimes terminate fatally in a few days or even hours. Such patients will usually give the history of shortness of breath on exertion and occasional præcordial distress of some yea- standing, while generally otherwise able to enjoy life. In the cases the failing power of the heart is due to degenerative chang in the heart muscle which suddenly gives way.

Finally, when the rare phenomenon of rupture of a valve se ment is the cause of the lesion, there is suddenly appearing dys nœa, præcordial pain and grave and general collapse. The patie may rally and live from a week to several months or longer. Dur this period the consequence of the valvular defect will be manife

As a natural result of disturbances of the circulation general n trition will often show signs of impairment. The patients ma become anæmic and pale and the mucous membranes blanche There will be coldness of the extremities, chilly sensations and d ficulty in keeping comfortably warm in cool, or cold weathe There is also a tendency to bronchitis and pulmonary disorde which are easily excited and intractable in their course. Som times these symptoms assume a severe character and, appearing a they do in association with the general malnutrition and wastir they may likewise excite the suspicion of tuberculosis.

Pulse.—The pulse will be one of the first features which w present itself for examination. Alone, it is not sufficient in an way to determine diagnosis, but in connection with other phenom ena it is of great importance. When compensation begins to fa it will show irregularities and intermittency, and in the later stag fluttering. In mitral insufficiency it may remain unaffected f years, but when changes appear it becomes small and weak, at th same time retaining its regularity. This is due to the left ventric losing blood by regurgitation and to the consequent diminution o the quantity of blood supplied to the aorta and arterial circulation In mitral stenosis, the pulse during the early periods of the diseas [illegible] with a low blood-pressure [illegible] vessel becomes mor [illegible] irregular and the [illegible] decreased blood- [illegible] in the left auri- [illegible]

[illegible] Here the blood [illegible] ventricle, then

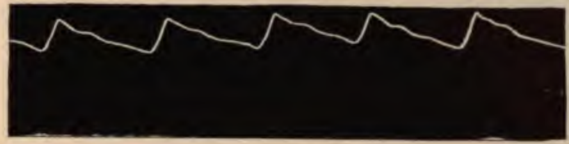

FIG. 10.—Normal pulse.

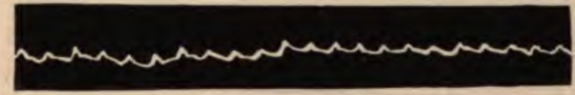

FIG. 11.—High tension pulse with nephritis.

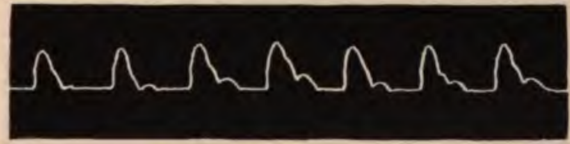

FIG 12.—Pulse of mitral insufficiency.

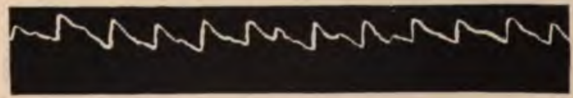

FIG. 13.—Pulse of mitral insufficiency with tricuspid insufficiency and failing compensation.

Actual sphygmographic tracings from cases in the Metropolitan Hospital.

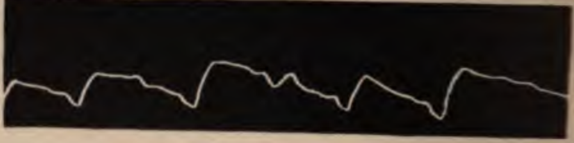

FIG. 14.—Pulse of mitral stenosis.

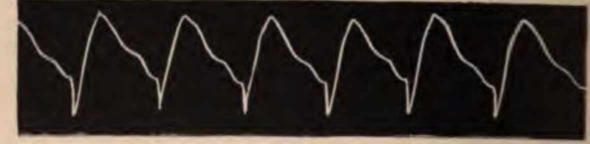

FIG. 15.—Pulse of aortic insufficiency and aortic obstruction. The former predominating. Acute benign endocarditis, second attack.

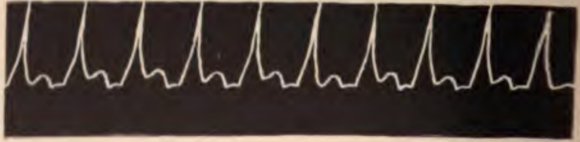

FIG. 16.—Pulse of aortic insufficiency with aortic obstruction.

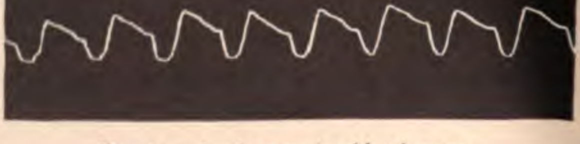

FIG. 17.—Aortic stenosis with atheroma.

Actual sphymographic tracings from cases in the Metropolitan Hospital.

falls back through the insufficient valves, each pulsation is abrupt and full, but of short duration, quickly falling away from the touch. In aortic stenosis the blood-pressure is variable, being sometimes above, sometimes below, normal. The pulse is usually slow and regular; it may be small, hard and incompressible. This is caused by the hypertrophied left ventricle propelling a smaller amount of blood than normal through the contracted aortic orifice.

Anomalous types of pulse may occasionally occur. The pulsus deficisiens with. mitral stenosis and insufficiency, and the pulsus bigeminus in mitral insufficiency have been observed.

It is important to note in this connection that lack of perfect rhythm is not necessarily an indication of cardiac disease, as it may occur in health when no abnormality of the heart or reflex influence is recognizable.

The Countenance.—The physiognomy may show changes which, when taken in connection with other symptoms, are more or less significant. Often it has a puffy, blanched appearance. In defects of the mitral valve it is apt to display evidence of venous congestion which is likely to be observed in cases of mitral stenosis, in those of combined mitral stenosis and insufficiency, and in the later stages of mitral insufficiency. In aortic disturbances there is a tendency to pallor and cyanosis. Changes of an icteric character may also appear and sometimes are very marked in connection with gastro-duodenal catarrh and hepatic congestion from venous stasis. They may also occur from absorption of coloring matter of the blood in pulmonary infarction.

Dyspnœa.—Dyspnœa is a symptom common to all forms of valvular defects, but is more marked in mitral than aortic disease. It is frequently one of the earliest manifestations of failing compensation, and is also present in combined lesions and all forms of heart failure. It is caused by stasis of the pulmonary circulation, whereby arises engorgement, and by diminution of the lumen of the alveoli from bulging of the walls of the capillaries. Thus there is not only stagnation of the pulmonary circulation, but also loss of surface in the air cells.

Dyspnœa always becomes aggravated when the recumbent posture is assumed, for the reason that that position places greater stress upon the weakened heart and pulmonary circulation.

Secondary Symptoms of Failing Compensation.—The conditions which secondarily arise in connection with interference with the normal action of the heart are of the gravest significance. They are the results of diminution of the blood-supply, stasis of the circulation and emboli.

One of the earliest manifestations of failure of the heart power is decrease in the quantity of blood supplied to the general system. Not only is there loss of propulsive force, but on account of insufficient filling the vessels will lose more or less of their resiliency. The effect of this ischæmia may show itself in all organs.

Furthermore, as a natural result of the obstruction of the onward flow of the blood, accumulation in the capillaries and venous system ensues; hyperæmia takes the place of anæmia and in time general stasis occurs. Under these conditions all organs may suffer from malnutrition and both functional and structural changes develop. Toxic material collects in the system and defective oxygenation will be induced by increased excretion of uric acid.

Dropsy.—When obstruction to the circulation has become sufficiently advanced to produce stasis, dropsy will manifest itself. This, as a rule, does not happen until the mitral and tricuspid valves have given way. Owing to the failing propulsive power of the heart the capillary vessels of the lungs become distended, the blood accumulates and is dammed back in the right side of the heart and stagnation of the general circulation of the venous system ensues. This interference with the return flow of the blood-current leads to increasing distention and finally to over-distention of the venous capillaries, until transudation into the tissues of the more watery parts or the blood takes place and the parts become œdematous and dropsical, as shown by pitting on pressure. Dropsy usually first makes its appearance at the ankles. The top of the foot will also show evidences of œdema. Later it will mount the leg, first involving the anterior tibial region, and gradually increasing. In time the œdema extends further upwards, and involves the thighs, abdomen and genitals. The penis and scrotum will become œdematous and translucent and the abdomen tense. Dropsy will also extend to the face and hands and the condition will become one of general anasarca. Owing to the distention, the skin will present a smooth, glazed, livid appearance and will feel waxy and cold. On account of the changed state of the circulation the skin not infrequently will become hypertrophied from hyperplasia of the subcutaneous connective tissue. In some instances the legs become so distended that the skin ruptures and a large amount of fluid exudes. On account of the low vitality dermatitis, erysipelatous inflammations and even gangrene of the skin may appear. Occasionally the œdema is more marked in one limb than in the other. In the case of the upper extremities, probably owing to the oblique course of the left brachio-cephalic vein, the dropsy appears earlier in the left than in the right side.

As the dropsy becomes general not only will the abdominal walls be loaded, but the peritoneal cavity will contain more or less fluid. Finally, fluid may accumulate in the pericardial sac, which in some cases will arrest the heart action. But more frequently patients suffering from valvular disease succumb to other complications. The most frequent termination is pulmonary œdema arising from the weakness of the heart, attended with extreme dyspnœa and cyanosis. In not a few instances the onset of the pulmonary œdema will be sudden.

In the later stages of cardiac dropsy the brain may show symptoms of disturbance; there will be confusion of ideas, dulness, lethargy, delirium and coma.

The distended limbs will become burdensome, while the effusion into the peritoneal cavity may increase so as to press up against the diaphragm and add further impediment to the already difficult respiration.

At this period pulsation of the jugular veins is not uncommon, as the tricuspid valves have become incompetent and the valves of the veins are unable to act, owing to the distended condition of the walls of the vessels. The pulsations of the right auricle may be transmitted to the neck. The cervical veins will also be distended, especially so during expiration, as their flow into the thoracic vessels will suffer more obstruction at that time.

Alimentary Canal.—Consequent upon interference with the circulation of the liver there will be symptoms of gastric disturbance, arrest of secretion, catarrh and symptoms of a so-called bilious character, constipation and hæmorrhoids.

Liver.—While the secondary effects of stasis necessarily are manifest in all organs to a greater or less degree, none are m affected than the liver and kidneys. Stagnation of the blood-f through the liver necessarily is attended with engorgement of t organ. Sometimes there is great increase in size and the marg may be felt on a line with the umbilicus.

In cases where obstruction in the hepatic circulation has be excessive and persistent, structural changes follow ; the organ b comes marked by a yellow and blackish deposit, the former bei due to fatty degeneration and the latter to stasis in the radicles o the hepatic veins. Mixed with these points of coloration there is general reddened line which arises from disorganized blood. Th internal structure of the line on section discloses a characteristi mottled appearance, known as the "nutmeg liver," a conditi generally regarded as pathognomonic of persistent stasis fro weakness of the heart action.

Perversion of nutrition may likewise lead to hyperplasia of th hepatic connective tissue, which in time undergoes contraction an cirrhotic induration, constituting what is termed cardiac cirrhosis i contradistinction to that of alcoholic origin.

When the tricuspid valve has become incompetent, pulsation o the liver may sometimes be observed anteriorly and laterally. In some instances it may actually be seen, while in others it can onl be felt on palpation.

Kidneys.—The changes which occur in the kidneys are similar t those which take place in the liver and constitute a serious complication. Distention of the veins will cause compression of th tubules and consequent structural changes. The urine will becom turbid, dark and scanty, and the amount will decrease as the blood-pressure is diminished. Examination will disclose the presence o albumin and casts and the general indications of a parenchymatou nephritis. Suppression of the urine may occur as a grave complication.

Spleen.—When there is considerable venous stasis and hepatic engorgement the spleen will become enlarged, but never to a great extent.

Nervous-System.—Nervous symptoms are frequently marked in the last stages, more especially in aortic lesions. There may be intense restlessness and irritability. Excessive blood-pressure may cause headache, vertigo, tinnitus aurium and muscæ volitantes. Attacks of syncope dependent upon cerebral anæmia may occur, especially in connection with aortic stenosis. In this condition, notwithstanding the hypertrophy, the constriction of the valvular orifice will not permit the normal amount of blood to pass through, and when the narrowing is excessive the brain will show symptoms of defective blood-supply; in consequence there will be attacks of faintness and vertigo.

Furthermore, as a natural result of obstruction, insomnia may be persistent or sleep may be broken and disturbed by dreams. Mental disturbances of a severe nature may manifest themselves. Cerebral hæmorrhage may occur especially in connection with aortic insufficiency, being favored by the high tension of the circulation. The membranes of the brain may suffer from venous congestion and embolism may prove the source of serious cerebral complications. Convulsions are rare, but may occur from concomitant uræmia.

The formation of embolic processes constitutes another division of the grave changes which are associated secondarily with chronic valvular disease. Here the thrombotic masses which are detached and thrown off into the circulation are generally more bland and of less frequent occurrence than in acute endocardial inflammation. The changes which are produced are mostly of a mechanical nature and may be regarded as true infarctions. Cerebral emboli are more frequently located in the fissure of Silvius as recognized by the sudden hemiplegia and aphasia.

When the lungs are the seat of embolic processes the symptoms may vary widely in severity. Some pulmonary infarctions may not give rise to symptoms, while, on the contrary, others may be the cause of immediate death. The first and most alarming symptom will be dyspnœa of a most urgent character, attended with great mental anguish and tumultuous heart action. Hæmoptysis may be present early. In mitral disease, especially stenosis, hæmoptysis must always be regarded as of important significance. Cough usually appears accompanied by expectoration of a dark, gelatinous, mucous character. In some cases shock will be profound, and when a main branch of the pulmonary artery has become occluded, or a large amount of lung tissue involved, there will be loss of consciousness and convulsions which speedily termina in dissolution.

Embolism of the spleen is recognized by sudden pain in the si tenderness on pressure and enlargement of the organ.

When the kidneys are the seat of lodgment the condition w also be marked by sudden pain in the affected organ, accompan by hæmaturia, cystic irritation and symptoms generally suggesti of renal calculus.

Emboli of the intestines, extremities and skin are also possib contingencies. Intestinal embolism may be suspected by the pre ence of pain, bloody diarrhœa, tympanites and vomiting. In th extremities, occlusion of the circulation at a certain point will giv rise to characteristic symptoms which are not easily mistake while multiple discolorations of the skin of a hæmorrhagic charac ter will show it to be the seat of embolic lesions.

Alteration in the quality of the blood does not appear to an extent in the earlier periods of valvular disease, but in the later stages when compensation has become much impaired changes ar more marked and leucocytosis and hydræmia develop.

Physical Signs.—From a diagnostic point of view, by far the most important symptoms of chronic valvular disease will be those elicited by physical examination. *Inspection.*—Following the time-honored order, the conditions observed by inspection will be thos first noted. The eye of the examiner will readily observe the general aspect of the patient. General dropsy or local œdema, cyanosis, labored breathing, as marked by the number of respirations and movements of the alæ nasi, paleness and anxiety of the countenance, will be recorded at a glance. Bulging of the chest walls over the præcordial region may be noticed in the young.

The impulse of the apex-beat is of the greatest importance as an anatomical point, and its situation is the first feature directly connected with the heart which the examiner should seek. In the majority of persons it is visible normally in the fifth interspace, about three and a half inches from the middle of the sternum, or just within the mammillary line. It is easily found in thin persons, while in the fleshy it is not so distinct. Any displacement shou at once be noted. In chronic valvular disease, owing to the hyp trophy present, the apex impulse will usually be displaced to t left and downward. The force of the stroke against the chest wall is in most instances slight and sometimes cannot be seen. On the other hand, it may be abnormally strong or intensified.

Palpation confirms and emphasizes the signs observed on inspection and is far more important. It determines the force of the apex-beat and the frequency or slowness, regularity or irregularity of the heart action. By placing the hand over the præcordial region the vibratory or purring thrill of a murmur or "*frémissement cataire*" of Corvisart may be felt. This occurs generally, but not always, in murmurs distinguished by their loudness and is most frequent in stenotic lesions, though it may occur in insufficiency, especially that of the mitral valve. The locality of the thrill, the time of its occurrence, and the relation it bears to the heart action in its different phases are points which should be carefully observed.

Percussion determines important signs, but it is not always possible to ascertain the outline of the heart with accuracy. It must be remembered that there are areas of cardiac dulness which include the locality in which the heart lies behind the chest walls without intervening organs, and that of superficial or relative dulness, in which the heart is covered by the thin layer of the borders of the left lung. The former is bounded as follows: On the right side, left sternal line; on the left, outside the left parasternal line; above, the fourth rib; below, the borders cannot be definitely made out, it being determined by the apex-beat and the upper border of the sixth rib.

In children the area of absolute dulness is greater on account of the heart being relatively larger. Here the upper border will be in the third intercostal space; the apex-beat in the fourth, and the left border near the mammillary line. In the aged the area is somewhat contracted from inflation of the lungs. During deep breathing the area of dulness becomes smaller, owing to the costal cartilages coming together at the sternum; quiet respiration has little or no effect. The dorsal or upright positions have no especial influence on the results obtained by percussion, but examinations made on the side will produce material changes.

Sometimes considerable tympanitic resonance will be elicited over a portion of the area of dulness. This will more frequently be observed in persons with a thick, short thorax and a large abdomen.

It is due to the stomach which lies behind the heart and is elici by strong rather than light percussion. When the heart and lu are normal it has no pathological significance.

The area of superficial or relative dulness is located to the l and above that of absolute dulness and corresponds to that porti of the heart which is overlapped by the borders of the lung. I will be revealed by stronger percussion. This region has be the subject of considerable discussion and there are decided diffe ences of opinion regarding its borders. Its clinical significan however, is secondary. The area may be considered as formin a border above that of absolute dulness commencing at the lowe margin of the third rib, continuing in a curve downward to th left within the mammillary line. In exceptional instances a poin of relative dulness may also be observed at the right of the abso lute dulness, limited by the inferior border of the sternum. I children this will be noticed to commence at the third intercosta space extending somewhat below the left mammillary line. It i also present on the right and may extend beyond the right side o the sternum.*

From a practical and clinical standpoint, the region of absolut dulness need only be taken into consideration as far as percussio is concerned. The heart may be regarded as enlarged when th dulness extends to or begins above the third rib, when it reache to or beyond the right border of the sternum, and to the left whe beyond the normal position of the apex. There is no lowe border as the dulness of the heart becomes blended with that of th liver. If the right ventricle is enlarged the dulness will be sprea toward the right, sometimes somewhat toward the left; if the le ventricle is involved the dulness is toward the left and downwards sometimes upwards also, but scarcely ever to the right.

The following, as given by Vierordt, is an excellent rule for percussion of the heart. On both sides of the sternum and close to it percuss strongly going downwards and observe the upper border of relative heart dulness; next percuss lightly the upper border o absolute dulness; then percuss lightly the upper border of the region of absolute heart dulness; next percuss upon the outer extremities of imaginary radii drawn from the centre of what is regarded as the region of absolute dulness, first the radius running obliquely upward and to the right, then from the right, always beginning beyond the sternum ; then on the radius running obliquely upward and to the left. Percussion should be strong at first to ascertain the relative dulness, then light for the absolute.

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* Medical Diagnosis, Oswald Vierordt, M.D., Philadelphia, 1891.

Auscultation is the most valuable and conclusive means of physical diagnosis. The best results are obtained when the patient is standing or sitting. Examinations also should be made when the patient is recumbent and the variations noted. Likewise when the patient is holding his breath and during natural and forced respiration. It is always best to examine the heart several times. It is likewise advisable to have the patient exert himself a little when practical.

Before considering abnormalities of the heart-sounds by auscultation it may not be amiss to review briefly the sounds of the normal heart. The natural heart-sounds are decidedly characteristic and can be heard over the whole præcordial space, but at different points they possess different features and different sources of origin.

The first sound is comparatively long and corresponds with ventricular contraction. It is therefore systolic. The second is short and abrupt and is heard with the diastole of the ventricle, and is consequently diastolic. After the second sound there is a pause. The sequence of the first and second sound and the period of rest constitutes the rhythm of the heart's action. The first sound is of a mixed character and is made up of sounds produced by the muscles, valves and vessels ; the second is wholly valvular from the action of the semilunar valves. The elements which enter into the production of the heart-sounds are chiefly the following : The sudden tension of the closure of the mitral and tricuspid valves, which is heard with greater intensity in the neighborhood of these valves and over the ventricles ; the closure of the aortic and pulmonary valves producing a diastolic, flapping sound, heard over the valves ; contraction of the ventricles, a sound of short duration ; and lastly, the sudden filling of the aorta and pulmonary vessels, which also cause sudden tension of these vessels and produces an abrupt sound.

The tones which are audible in health correspond with the sounds of the heart, as follows :

APEX OF THE HEART (Mitral orifice).

First sound: Closure of the mitral valve and contraction of the ventricles.

Second sound: Prolonged aortic second sound, closure of the aortic valve.

UNDER THE STERNUM (Tricuspid orifice).

First sound: Closure of the tricuspid valve and contraction of the ventricles.

Second sound: Prolonged pulmonary second sound.

SECOND INTERCOSTAL SPACE, RIGHT OR LEFT (aorta and pulmonary artery).

First sound: Sudden filling of the aorta, of the pulmonary artery, continuation of the first ventricular sound.

Second sound: Closure of the semilunar valves of the aorta or of the pulmonary artery. (Vierordt.)

For anatomical reasons the valves which are most important from a clinical standpoint, namely, the mitral and aortic, are not heard to the best advantage at the areas which lie directly over them. In the instance of the mitral valve this is due to the enveloping of the heart by a layer of lung at the point where the valve is located. The mitral sound is heard best at the apex. In auscultating the aortic valve we must listen at the commencement of the aorta, for the reason that the aortic valve lies obliquely behind the pulmonary, the sound of which predominates.

The pathological processes which change the structure of the valves and orifices obviously affect the flow of blood through the heart and produce alterations in the normal sounds. Lesions, however, may exist, as previously stated, without apparently interfering with the action of the valves, but, as a rule, valvular disease affects the normal heart-sounds in many ways, and is attended with alteration in intensity, quality, pitch, rhythm and the development of adventitious sounds. The recognition of the existence of these sounds, or, as they are generally called, murmurs, will determine definitely the presence of valvular disease. Generally, when the heart-sound is distinctly heard in conjunction with the murmur the esion is not extensive, when, on the other hand, it is lost, being entirely replaced by the murmur, it is severe.

Adventitious heart-sounds will present many variations. Some will be soft and blowing, and have been aptly compared to the word "who" when whispered, and are heard mostly in insufficiency; others are of a creaking, grating nature, while others again may be suggestive of a musical tone. They will also vary in intensity; some can scarcely be heard, while others are exceedingly loud. In cases of the former type exercise may render the murmur more distinct, and even make apparent those which could not be heard before. The duration of the sounds will be equally variable; when damage to the valve has been extensive and is of the nature of a rupture of a segment or tendinous attachment, the murmur may be of peculiar, almost screaming, character. This is the "*bruit piaulement*" of the French.

Murmurs being the result of changes in the structures of the valves or orifices are necessarily associated with the phases of the heart action; thus they may replace the sounds of the normal heart or may precede or be appended to them. Therefore, as they are observed in relation to systole or diastole, they are termed systolic, diastolic, presystolic, and subsystolic. When the heart is beating regularly or comparatively so, it is not difficult to determine whether the murmur is systolic or diastolic, but when its action is tumultuous, feeble or irregular it is difficult and sometimes impossible. When a mitral murmur follows the first sound after an appreciable interval it is called "retarded" and signifies that the valves close, yet are unable to remain in perfect adaptation to the orifice during contraction of the ventricle and thus leakage ensues. The danger under these conditions may be considered slight. A "retarded" aortic murmur may be encountered, but it does not possess the same favorable indication.

The extent of area over which a murmur may be heard is another important feature and one also subject to wide differences. Some will only be detected in the region of the valve or orifice which is defective, while others may be audible a long distance from their seat. In all cases of a widely dispersed sound the point of greatest intensity can be located and the seat defined. It will thus be observed that the two points of importance in this connection are the seat of maximum intensity and the sound of the heart with which the adventitious sound is synchronous.

In considering chronic valvular lesions, as the left side of the heart is the chief subject of study, the mitral and aortic valves with their orifices will demand almost exclusive attention.

Mitral lesions are heard with the greatest intensity at the apex. Position is of importance. When the patient is upright or leaning forward the sounds are naturally more readily distinguished. Aortic lesions are best observed at the junction of the second rib with the sternum on the right side. In some cases they are difficult to hear and can only be detected by change of position. There are certain variations of the strength of the heart-sounds within the limits of health. These differences will depend upon the elasticity and delicacy of the thorax and upon the thickness of the chest walls. Accumulations of fat and large mammæ naturally weaken the sounds. In children and persons in whom the thorax is delicate the sounds are loud. Excitement may also increase the intensity of the heart-sounds and it must be remembered that in some cases the mere fact of an examination may have a disturbing effect on the heart action. The rhythm of the heart-sound also varies within normal limits.

While lesions of the right side of the heart are rare, an exception must be made in the case of the tricuspid orifice where relative insufficiency is quite common. This arises from the frequency of change in the size of the right heart. The presence of tricuspid insufficiency indicates dilatation of the right ventricle. Lesions at the tricuspid are heard to the best advantage at the junction of the fifth and sixth left rib with the sternum. On account of dilatation the position of the valve will become changed so that the sound of insufficiency may be heard with intensity as far down as the ensiform cartilage where the pulsations of the ventricle may be clearly seen.

In the case of the pulmonic valves direct lesions are of such extreme rarity that they may be almost excluded from consideration. At the same time the second pulmonic sound is of great importance, as it is an indicator of the action of the right ventricle as the pulse is of the left. The pulmonic valve-sounds will be heard to the greatest advantage at the base of the heart at the junction of the second left rib with the sternum.

Alteration in the rhythm of the heart may be observed in the relation of the first beat to the second and in that of one cardiac rhythmic cycle to another. Simple hypertrophy with pulmonary congestion may cause a succession of strong beats followed by a number of feeble ones, uniform or nearly so. When hypertrophy and dilatation are attendant upon mitral insufficiency, time and force are both profoundly affected. In the case of its association with aortic disease, force more than time is affected. Great irregularities attend excessive dilatation, fatty degeneration and malformations.

Variations in the quality of the first sound belong to both health and disease. Normally in thin, nervous persons, it is high pitched, short in duration and ringing in quality; in vigorous persons it is lower, longer and more rumbling; in the stout and indolent it is feeble and short.

In mitral obstruction the first sound is frequently intensified, while in insufficiency it is often the reverse. Increased intensity or loudness from hypertrophy of the left ventricle with renal cirrhosis might be expected, but the sound is often low, long and muffled. In febrile diseases when there is increased arterial pressure with some excitement of the heart there may be some increase of intensity of the first sound. In aortic disease the first sound is sustained, low and rumbling. This is commonly heard in hypertrophy.

Changes in the first sound are also important. *Reduplication at the apex* indicates absence of perfect synchronous action of the two ventricles and may occur when undue pressure is put upon the left ventricle by high arterial tension, or upon the right by obstruction in the pulmonary circulation. Thus it may be observed in advanced aortic disease, or in other conditions associated with high arterial tension, such as nephritis, where it indicates that the left ventricle is beginning to weaken. In the case of the right ventricle the phenomenon may occur when there are obstructive lesions of the lungs. According to Gibson, of Edinburgh, the doubling of the first sound is more apparent than real, and all that really occurs is that the maximum intensity of the left first sound is not coincident with the maximum intensity of the first right.

Decrease of intensity of the first sound of the left ventricle is heard in the later stages of mitral insufficiency and of fever, and in degeneration of the heart wall.

Intensity of the first sound of the right ventricle over the tricuspid area may vary greatly. Increase is not so often observed for the reason that when obstruction of the left side leads to increase of pressure within the right, with pulmonic accentuation and increase of the tricuspid first sound, after a certain limit the safety-valve action of the tricuspid comes into play and the increased pressure is diminished.

Decrease of intensity of the first sound in the tricuspid area is heard in all states of the circulation in which there is enfeeblement of the right ventricular wall from any cause—pyrexia, degeneration, malassimilation, etc.

Normally the second sound is heard at the base more distinctly than the first. If the contrary condition exists, the second sound is abnormally weak.

Reduplication of the second sound at the base may be heard both physiologically and pathologically. Various explanations have been offered for its causation, that generally accepted being difference of pressure in the aorta and pulmonary arteries whereby the aortic and pulmonary valves fail to close in unison. Sanson explains the phenomena as due to sudden tension of the abnormal mitral curtain from relaxation of the wall of the ventricle. This, however, offers no clue for the production of the double sound in health. Pathologically, the doubling of the second sound is heard in mitral stenosis and indicates that the pressure in the pulmonary circulation has increased to such an extent as to cause the pulmonic valves to close before the aortic. Physiologically, it may be observed at the end of inspiration and on holding the breath after a deep inspiration.

Changes in the intensity of the aortic second and pulmonic second sounds are of important significance. When it is desired to estimate the relative intensity of the pulmonic valves, the aortic valves must be considered in comparison. The pulmonary second sound may be distinguished from the aortic by the fact that it is heard only in a small area, namely, at the second left interspace near the sternum, while the aortic valve may be heard over an extensive area including the apex. Normally the pulmonic sound is louder than the aortic, hence it follows that if the two sounds are of equal intensity, the aortic is accentuated.

Accentuation of the aortic second sound indicates a condition of high tension in the arterial system. It is observed in fusiform, globular and sacculated dilatation of the aorta (aneurysm), hypertrophy of the left ventricle, especially in association with thickened and contracted arteries, disease of the kidneys, atheroma and sclerosis, and in all conditions of abnormally high arterial pressure. In hypertrophy of the left ventricle with aortic insufficiency, accentuation is absent, for the murmur either displaces the second sound entirely or in part. Not that it overpowers it, but the accentuation is enfeebled or lost on account of the insufficiency of the valves.

Diminution of the second aortic sound signifies enfeeblement of the wall of the left ventricle and is observed in mitral obstruction, pulmonary affections in which the return of blood to the left ventricle is impeded, and in all conditions which tend to weaken the wall of the left ventricle, such as degenerations, pyrexia and relaxed conditions of the arterial system. Accentuation of the aortic sound, therefore, in valvular affections, implies strength of the left ventricle, while diminution implies the reverse.

Accentuation of the pulmonic second sound denotes increased pressure within the pulmonary circuit and arises from causes which induce obstruction of the blood-current through the lungs, as in emphysema, consolidations, fibroid induration and in mitral disease, both regurgitative and obstructive, always supposing the right side of the heart to be intact. In mitral regurgitation the pulmonic sound is accentuated because the left ventricle during contraction drives back a certain amount of blood through the imperfect mitral valves into the left auricle, and in consequence the pressure in the pulmonary circuit is increased and accentuation results. In mitral obstruction pulmonic accentuation is produced by retention of blood in the left auricle, which arises from the obstruction at the mitral orifice and which exerts an opposing force to that of the right ventricle. The blood under these conditions is under an abnormal state of tension and the pulmonic valves put upon a strain. In mitral disease accentuation of the pulmonic second sound signifies hypertrophy of the right ventricle and severity of the lesion ; at the same time it denotes strength of the right ventricular walls.

Diminution of the second pulmonic sound means dilatation and weakness of the right ventricle and is therefore of grave significance.

It is observed in advanced forms of mitral disease and in conditi of failing heart power. It might be expected to occur in tricusp insufficiency, but this is seldom discoverable.

Conditions of combined murmurs are very frequently observe Such lesions are said to be double. Both insufficiency and stenos may be present. The sounds vary in character and intensity a cording to the nature of the lesion, and the same observations appl as in the case of single lesions.

Diagnosis.—The diagnosis of cardiac murmurs will be considered when treating of the different varieties of valvular affections.

Prognosis.—As the numerous factors which enter into the æt ology of cardiac disease necessarily all exert a strong influenc upon the prognosis, the future of each case must be viewed from many standpoints. It is of the utmost importance that the probable cause of the disease should be known and whether or not a sudden termination may be looked for. It is also essential that the patient's fears should be allayed as far as possible, as apprehension will tend to aggravate the trouble.

The influences which must be considered in determining the prognosis of valvular disease may be summarized as follows: (1) The nature of the lesion; that is, the valve involved and the extent to which it is affected. (2) Whether the lesion has a tendency to remain quiescent or otherwise. (3) The general condition of the heart. (4) Age of the patient. (5) Occupation and habits of living. (6) Heredity. (7) The influence of complicating disorders.

There is a class of cases where the patient has continued in apparent health for a long period with little or no inconvenience or disturbance and no symptoms but the murmur; there is no change in the pulse, very little hypertrophy or dilatation, while the murmur will seem more to accompany the heart-sound than to displace it. Under such conditions there is no present danger, and if the lesion has already been of long standing, and especially if it has arisen from acute rheumatic endocarditis, the chances of a long life are very favorable.

There is another type of cases similar to the above as to the absence of secondary symptoms and freedom from disturbances, yet which differs in that dilatation and hypertrophy are very marked. In this instance, while compensation is sufficient, the valvular defects and myocardial changes are so decided that the equilibrium of the circulation is easily deranged. Under these circumstances, while the patient may remain free from disturbances, any intercurrent disease, especially of the lungs, will be attended with gravity.

Still another type of cases is observed when the symptoms of failing compensation are beginning to appear. There will be palpitations, shortness of breath on slight exertion, oppression and pain in the præcordial region, slight œdema of the feet and ankles, thick reddish deposits of urates and albuminuria. In such conditions danger is impending, though life may be prolonged for a considerable period by careful treatment.

In advanced cases of valvular disease when serious symptoms have developed the outlook is decidedly unfavorable. If the lesion has been aggravated by some intercurrent affection, such as bronchitis, debility or anæmia, the possibility of relieving the condition will render the prospect of prolonging life more favorable ; but in all cases with general anasarca, pulmonary congestion, dyspnœa, inability to lie down on account of oppression, and the presence of renal complications, there is little hope and the end is not far.

Great differences exist in regard to the relative gravity of the several lesions. Aortic insufficiency is the most rapidly fatal when it occurs late in life, as compensation is then established with difficulty. In childhood and early adult life mitral stenosis is frequently more serious than aortic incompetence, owing to the progressive nature of the disease. Generally speaking, aortic stenosis is attended with less serious consequences than mitral stenosis, and aortic insufficiency is much more dangerous than mitral insufficiency. While there is some difference of opinion in respect to the relative dangers of the different varieties of lesions, the following order in regard to their gravity seems that the most generally accepted : Aortic insufficiency, mitral stenosis, aortic stenosis and mitral insufficiency. While investigations of statistics bear out this enumeration, the practical value of any such estimate must be of limited utility, owing to the fact that there are many influences which bear strongly upon each case and which, not infrequently, are of more importance than the mere question of the variety of lesion.

The question of sudden death, which in the minds of the laity is intimately connected with valvular lesions, is one which the physician will be called upon to answer. By sudden death is not meant the abrupt termination of a case with non-compensation and secondary symptoms, but sudden dissolution of a person in apparently fair health. It is generally conceded that the form of valvular disease wherein this contingency is most likely to occur is aortic insufficiency, though Stokes maintained that mitral disease was most prone to end in sudden death.

The extent of a lesion is a very difficult matter to determine. Physical examination alone does not reveal the exact condition. The character of the murmur, the pulse, the amount of dilatation and hypertrophy will serve somewhat as an index of the extent of damage to the valve. The dilatation may be so small as to be scarcely capable of detection, or it may be excessive. In the first instance the defect in the valves will be slight, and in the second extensive; thus an important factor will be ascertained which must be considered in its relations to treatment as well as to prognosis.

A murmur associated with dilatation which tends to disturb compensatory hypertrophy or shows any tendency to asystolism is grave. Manifestations of disturbance in other organs, as the stomach, kidneys or liver, secondary to valvular defects, are evidences of serious significance.

The tendency of the morbid process to progress is obviously an element which must greatly influence the future of each case, and one which must always be taken into consideration. Remembering that the causes of valvular diseases are the sequelæ of acute endocardial inflammation, chronic endocardial inflammation, degeneration of the valves and myocardium, and mechanical influences, the question as to the character of the changes will not be difficult to answer. If the cause be acute endocarditis there is good reason to suppose that the lesion once developed will remain stationary after the subsidence of the acute conditions. An exception as stated elsewhere will be observed in the instance of mitral stenosis, where the morbid process is progressive, the valve gradually becoming narrower, owing to continuous contraction of the cicatricial tissue.

In the instance of chronic endocarditis and degenerations an entirely different type of morbid influences is encountered. Here the lesions are essentially those of the later periods of life and are characterized by their progressive nature and generally unfavorable aspect and insidious onset. When an acute endocardial inflammation is the cause of the lesions it is not difficult to arrive at a conclusion as to the time when the valvular integrity was primarily damaged, but it is impossible to state with any degree of accuracy when a chronic inflammatory or degenerative process first located in the cardiac structures. While these conditions are all prone to be progressive, their course is characterized by marked differences; in some cases the changes will develop rapidly, while in others they are slow in manifesting themselves. Compensating dilatation will follow and the heart will be able, to a greater or less extent, to adapt itself to the altered conditions, but compensation, as a rule, will eventually fail. For not only are these pathological processes progressive, but they are operative at a time of life when the heart has less power to accommodate itself to new conditions and is itself subject to structural degenerations.

There are, however, conditions which qualify this unfavorable view, for valvular lesions are encountered in middle and later life which are not apparently traceable to any acute endocardial inflammation and which arise from some chronic morbid process, yet beyond the physical signs are not attended with disturbances. In these lesions, the processes, while not stationary, are decidedly slow in their course, and allow the patient to enjoy many years of comparative comfort; at the same time it must be remembered that as age advances the condition is liable to become aggravated, and if an intercurrent disease appears the valvular lesion is liable to add serious complications.

The prognosis in cases which are induced by mechanical influences will depend upon the cause. Degenerations arising from strain will follow a course similar to that produced by other influences. Rupture of the valve is a condition of such rarity that any generalization of cases is difficult. In some instances death is instantaneous; in others, after the subsidence of the acute condition, it may be followed by secondary symptoms, the same as in advanced cases of other grave forms of heart disease.

The influence of age has already been alluded to. In very young children the development of valvular defects is always attended with danger, from the fact that acute rheumatic endocarditis, which is generally the cause, is liable to return and commit further ravages, and in some instances so damage the heart as to exercise deleterious effect upon the nutrition and growth of the child. The influence of old age upon the course of heart disease is characterized by difficulty in the establishment of compensation, and the tendency of the degenerations peculiar to the period of life to undergo fatty and fibroid changes.

While sex cannot be said to have much effect upon prognosis generally speaking, compensatory dilatation and hypertrophy are more readily established in males than in females.

Heredity must be taken into consideration when giving an opinion in regard to the future of valvular affections. The diathetic conditions which prevail in a family, whether or not its members have been long-lived, and especially if cardiac lesions have been prevalent to any extent, are factors which will bear strongly upon the future. The conditions which seem more under hereditary influences are those connected with degeneration of the heart walls. High arterial tension in itself is frequently hereditary and will greatly aggravate the existing conditions and exert an unfavorable influence upon the prognosis.

Occupation, habits and manner of living are elements which must always be regarded. Cases of valvular lesion in persons who are compelled to engage in manual labor, especially of an arduous character, must obviously run a shorter course than in those more advantageously placed in life. Bad hygienic surroundings and exposure to the inclemencies of weather and extremes of temperature will tend to weaken the efforts of compensation. Those who, in addition to being compelled to engage in laborious tasks, are ill-fed, ill-clothed and ill-housed, and perhaps given to excessive use of alcoholic beverages, are clearly under influences best calculated to hasten the degenerative processes. Compensatory hypertrophy has a poor chance under such conditions. On the other hand, habitual persistence in immoderate eating and drinking will increase arterial tension and augment the influences which are operative in causing the morbid conditions, and necessarily exert a deleterious effect.

The presence of complications, especially anæmia and pulmonary affections, will augment the danger. The effect of anæmia upon the structures is decidedly deleterious, being in itself capable of giving rise to decided disturbances of the circulation; when associated with a valvular defect it will greatly aggravate the situation by inducing dilatation of the left ventricle and regurgitation at the mitral orifice, thus increasing the dilatation. It will, in addition, impede compensation by impoverishing the blood and general nutrition.

Treatment.—Chronic valvular lesions, unattended with disturbing phenomena, do not call for therapeutic interference. On the other hand, when the source of consecutive symptoms or in association with other morbid conditions, careful treatment will be urgently demanded.

Prophylactic measures are of limited practical application, since the majority of cases do not come under medical care until the affection has been long established and is beyond the question of prevention. When, however, there is reason to apprehend that the valves will become damaged as the result of the presence of conditions liable to induce organic change in the endocardial structures, especially acute rheumatism, prevention is a matter of importance. Under these conditions the heart should be carefully watched, and at the first intimation of endocardial implication every effort should be made to avert the complication or, at least, to limit its extension. With this end in view the preventive measures mentioned in the article on pericarditis should receive consideration.

The earliest symptoms of cardiac involvement, it may not be amiss to recall, are alterations in the rhythm of the heart with accelerated, uneven and tumultuous action. When an acute inflammation of the endocardium has subsided and a valvular defect remains, great care is essential in order to prevent undue dilatation of the heart walls. Here rest, as stated elsewhere, is also of paramount importance. The patient should be kept in bed or confined to his room for several weeks or even months, according to the nature and severity of the lesion. Cases of aortic insufficiency will require a longer period of rest than other varieties of valvular disease. When pericarditis has been associated with acute endocardial inflammation, the period of repose should be of greater length, as the heart walls are much weakened.

Again, in persons suffering from diathetic conditions prone to induce cardiac diseases, especially those whose family history shows a tendency to valvular affections, the heart should be watched for evidences of insidiously developing lesions, and the use of alcohol, indulgence in dietary excesses and the effects of muscular strain must be taken into account.

The general management of valvular affections will vary according to the adequacy or inadequacy of compensation. The natural division separating these two classes of cases is broad and distinct. In those where, other than the physical signs of a murmur, there are no symptoms and evidences of hypertrophy and dilatation are absent, little need be said as to treatment; care, however, is needful. All influences which may in future bear upon the condition should be considered. Violent muscular efforts and occupations calling for great strength and endurance must be avoided, but exercise of many kinds may be enjoyed, for in numerous instances the presence of a valvular defect is not incompatible with a considerable amount of effort. Exercises, however, should be in moderation and within healthful limits. In the young, outdoor sports, such as tennis, golf, moderate rowing, riding and even dancing, swimming and bicycling may be allowed. In regard to the last special caution is called for and up-hill riding and long rides should be condemned. Foot-ball, running races, paper chases, violent gymnastics and exercise which require fixation of the chest and prolonged closure of the epiglottis should be prohibited. Exposure must be avoided and, in the case of rheumatics, flannel underwear should always be worn.

In patients in whom dilatation and hypertrophy are decided yet compensation remains adequate, the changes in the heart wall add a more serious aspect to the situation. Greater care is imperative lest the compensation be further weakened or destroyed. Such persons need not be considered as invalids as some would have, yet their future life must be more or less modified according to the demands of the diseased condition. In laying down the rules for living for this class of cases it should be the aim to interfere as little as possible with the ordinary vocation.

Those who are fortunate enough to be able to consider environment should reside in some mild, equable climate where the soil is sandy and the vicissitudes of temperature are at a minimum, for it must be borne in mind that many of these patients are of a rheumatic diathesis and are especially susceptible to climatic influences. High altitude should be avoided, one of 2000 to 3000 feet will be most suitable; more than that is not to be commended. Residence by the sea will prove of benefit in many instances.

The diet should be regulated. Excesses of all kinds should be avoided. When the arterial tension is high a hearty meat diet should be prohibited. Milk, eggs, green vegetables, fruits and the lighter varieties of meat should be favored, while the carbohydrates should be used in small quantities. The amount of liquids should be limited to actual requirements, since excess of fluid or of food tends to augment the work of the heart. Tea, coffee and tobacco should be prohibited, and alcohol should be used only under medical supervision. A small quantity of wine may frequently be taken with advantage with the meals, and if there is sleeplessness a little spirits in hot water at night time.

One of the most important questions in this type of cases is that of exercise. The patient should be instructed as to the necessity of avoiding violent muscular efforts or putting an undue tax on his strength. Many conditions of compensation which have remained adequate for a long period have been irretrievably damaged by a mountain climb or some such exertion. Those who are compelled by necessity to engage in manual labor of an arduous nature must seek, as soon as possible, occupations which do not call for continuous muscular strain. In all instances open air life should be followed as far as practical. Definite rules, however, are not possible; each case must be considered individually, the seat and character of the lesion, the age and temperament and sex of the patient must all be regarded as factors. Ordinarily what would be allowed in youth would scarcely be admissible in middle life. In the young, even in cases with some dilatation, a certain amount of outdoor sport may be permitted. Temperament also is important. Some are very timid and apprehensive; such must be encouraged. Some, on the other hand, are rash and impulsive and must be restricted accordingly.

A rule in regard to exercise in cases of valvular lesions, which is always applicable and which has no exception, is that fatigue should be avoided and that any sign of painful breathlessness should be regarded as a positive signal for rest. Furthermore, whenever participation in any particular form of exercise is attended with embarrassment of the respiration, it is a positive indication that such efforts are decidedly contraindicated. *Walking against a strong wind should be strenuously avoided by all those suffering with cardiac disease.*

In those cases characterized by failing compensation therapeutic measures are of the greatest importance. Here the disturbances will present types varying from the earlier periods of failure as evidenced by the attacks of palpitation and præcordial distress, dyspnœa on exertion, and slight œdema, to the advanced cases with general anasarca and nephritic complications. In the less severe forms of this type, and in those where the equilibrium of compensation is maintained with difficulty, much can be done to relieve the patient and to prolong life.

All that has been said in regard to climate and freedom from exposure holds under these conditions with renewed force; a mild climate should be selected and the patient enjoined to live in the open air as much as possible. All active exercise must be prohibited, at the same time total inaction will not be called for. The patient may be allowed to walk in moderation, but never when there is wind, or the temperature is very cold. The diet should be carefully supervised and food difficult of digestion, and overloading the stomach, must be avoided, as gastric disturbance may induce severe and alarming symptoms. The excretions should also be carefully observed.

As evidences of inadequacy of compensation begin to manifest themselves absolute rest will prove of great benefit in toning up the failing powers. The patient should be placed in bed and should remain there for a period varying according as the condition demands. The importance of this measure cannot be too strongly emphasized. The writer has seen many cases in which the evidences of failing compensation have disappeared under rest. At the same time, as the nutrition is low, a liberal diet should be administered consisting of frequent light meals of easily digested food. General tonics will often be indicated.

The selection of remedies in the treatment of valvular disease will be guided by the fact that decrement of arterial pressure is the great underlying factor in the symptomatology of the affection. The chief indications are those derived from observation of the general symptoms, the sounds and movements of the heart, condition of dilatation and hypertrophy, and the phenomena connected with the state of the pulse. It is needless to say that the action of remedies is on the heart muscle and not on the valvular defect.

Digitalis is the first remedy to suggest itself. Its utility in valvular disease is unquestionable. Its chief indications are weak, irregular, intermittent, rapid pulse; every movement causes it to beat faster, also slow pulse; pulse of low tension; sensation as if the heart stood still; dyspnœa; cough; dusky hue of the countenance; pulsating jugulars; scanty, high-colored urine; urine alternating scanty and copious; albuminuria; jaundice; hæmoptysis; œdema of the feet and ankles; general anasarca; vertigo and amaurosis. It is contraindicated when arterial tension is high and the action of the heart strong. Fatty degeneration and atheroma of the myocardium are not necessarily contraindications, but evidences of their presence should be regarded as a signal for caution.

Its sphere of action will be found principally in disease of the mitral and tricuspid areas accompanied by engorgement of the venous circulation. In mitral insufficiency it lengthens the diastole and thereby permits more blood to flow from the auricle into the ventricle. It also strengthens the systole and induces more complete closure of the valvular segments and in consequence less regurgitation. More blood is also propelled into the arteries. In mitral obstruction the lengthening of the diastole allows more blood to flow through the contracted orifice and thus affords relief to the engorgement of the venous system, but in the earlier periods of this lesion it is generally condemned. In affections of the tricuspid the action of the remedy is similar.

In aortic diseases the action of digitalis under some conditions is beneficial, while under others it may prove injurious. In the instance of aortic obstruction the left ventricle may be injured by driving the blood through the narrowed orifice, but when compensatory hypertrophy is absent and the heart action is rapid and feeble, or when it is desirable to strengthen the systole, the administration of digitalis will be attended with benefit. In insufficiency it was condemned by Corrigan on the supposition that the prolonged diastole allowed a greater amount of regurgitation. This theory at the present time is not regarded as substantiated, and the remedy will often be demanded in aortic insufficiency whenever there are signs of failing heart power with secondary involvement of the mitral valve. In aortic obstruction and insufficiency associated with dilatation and mitral regurgitation digitalis will also produce marked results. The tendency of digitalis to increase the resistance of the peripheral circulation and thus throwing more work on the heart must always be taken into consideration. Although the contractile energy of the heart under the influence of the drug may more than counterbalance this effect, it may, on the other hand, be unable to do so. When such is the case nitroglycerin should be administered in association with digitalis in order, by its action as a vasomotor dilator, to dilate the peripheral vessels and thus facilitate the onward flow of the blood. This combination of nitroglycerin and digitalis is of the greatest importance here. When it is determined upon, one drop of the 1 per cent. solution or of the second decimal dilution of nitroglycerin may be given with the desired dose of digitalis.

Strophanthus is probably one of the most useful substitutes for digitalis ; it may succeed where the latter has failed, although it is by no means its equal. It has the advantage of being free from causing gastric irritation, and may be administered in all forms of valvular lesions where compensation is lacking, being especially useful in mitral stenosis without degeneration of the cardiac muscle, and in cases of failing heart in cardio-vascular sclerosis associated with interstitial nephritis. On account of its property of not contracting the arterioles it will prove of value in cardiac dropsy with pulmonary and nephritic congestion. Its chief use is that of an adjuvant to digitalis and as a substitute in those cases where, notwithstanding the presence of characteristic symptoms, digitalis has proven ineffectual. Its action being more rapid than that of digitalis and its effect less lasting, the dose should be sooner repeated. It should never be given hypodermically, as it is liable to cause abscesses at the point of injection when thus administered. Care must be exercised to obtain a reliable preparation, as some are worthless.

Caffeina in valvular affections may be employed upon the same indications as digitalis, bearing in mind that its action is more rapid and much less lasting. It may be used when the latter proves irritating to the stomach and when decided diuresis is desired. In valvular disease, dilatation with or without fatty degeneration, it strengthens the heart and often produces excellent effects. It is well to commence with moderate doses and rapidly increase.

Sparteinæ sulphas has proved beneficial in cases of enfeebled heart with valvular deficiencies, especially in disease of the mitral valve when there is marked dilatation. Germain Sée recommends it in aortic and mitral insufficiency and mitral stenosis, the best results being obtained in the failing heart of mitral stenosis and aortic regurgitation. In insufficiency of the aortic valve with rapid, tumultuous action of the heart, it has proved itself effective. Small doses appear to act best.

Spartein may be used as an adjuvant to digitalis. Being more speedy in effect, it may be administered first to begin the action.

Adonis vernalis, like most of the drugs of lesser importance which compose the digitalis group, has no special individual indications. It has been employed with good results in cases of mitral and aortic regurgitation when digitalis has failed to act. The infusion or glucoside is recommended by Armstrong in cases of valvular disease with insufficient compensation attended with marked circulatory disturbances and in cardiac asthma. In valvular diseases of the heart arising secondarily, chronic nephritis with enfeebled heart action, irregular or intermittent pulse and venous stasis, it has in some instances produced very satisfactory results.

It is preferred by some observers, in place of digitalis for the failing compensation of mitral obstruction. A tolerance of the remedy will be experienced in a few days and the dose, therefore, must be increased, as with arsenic. It may also act as a purgative.

Cactus grandiflorus, while decidedly a remedy of secondary importance in valvular disease, is sometimes of great value, especially when its characteristic symptomatic indication is present, namely, "a sense of constriction as if the heart were being tightly grasped or the chest were bound by an iron band," when it will afford relief. It fails in feeble heart action dependent upon extreme dilatation and in mild obstruction, but is useful in aortic regurgitation.

Glonoinum (nitroglycerin or trinitrin) is indicated in all forms of valvular disease attended with high arterial tension and spasmodic contraction and rigidity of the vessels. Its special indications are :

Pain in the region of the heart radiating towards the back and shoulders, spasmodic and pressing sensation in the heart, violent cardiac contractions, slow beating and pulsation of arteries, quickened heart action, full, strong pulse, small pulse, thready and irregular pulse, pulse of high tension. It is of use in affording relief to the left ventricle by allowing more blood to flow to the peripheral vessels. In valvular defects associated with arterio-sclerosis, especially aortic insufficiency with angenoid pain, and in dilatation with degeneration, it is valuable. Cardiac asthma and severe dyspnœa are often relieved by it. Its administration in conjunction with digitalis to overcome the tendency of the latter to cause contraction of the peripheral vessels, to which allusion has been made, constitutes a most important feature of its application in valvular diseases.

Strychnia, in addition to its efficacy in sudden heart failure of all forms, is useful in valvular disease for steadying the heart, and as a general tonic to the muscular system. It may be administered in association with other remedies.

Potassii iodidum, *sodii iodidum*, *strontii iodidum*, *arsenici iodidum*, *aurum*, *baryta carbonica*, *barii chloridum* are all indicated in these forms of valvular disease associated with arterio-sclerosis. Their use must be continued for a long period.

Treatment of Special Symptoms.

ANÆMIA.—The anæmia which frequently accompanies cardiac lesions should be treated on the same general lines as under other conditions. Careful diet with blood-making foods and iron in some form are the chief resources. The tincture of the muriate of iron, the second decimal trituration of ferrum reductum, or of the arsenite or iodide, or Flint's chalybeate pill, will be found useful. In severe cases arsenic will meet the condition in the second decimal triturate of arsenicum album in two grain doses, of Fowler's solution of arsenic three times a day. Frequently iron and arsenic may be combined to great advantage. In these conditions a practical method of treatment would be the iron or iron combination after meals and the cardiac remedy every two hours between meals, or three or four times a day, as called for. Massage and faradization assist in some cases.

Angenoid Pains.—Aortic insufficiency with sclerosis is especially liable to develop these symptoms, although they may appear in other forms of valvular affections. When arterial tension is high nitroglycerin will prove useful. Arsenicum, spigelia, cactus, kalmia, lachesis, rhus, crotalus and aconite will also be effective, according to indications. When the pain is severe heat or cold to the præcordium, amyl nitrite inhalations and morphine or atropine hypodermically, as mentioned in angina, should constitute the line of treatment. If the heart action is slow or irregular diffusible stimulants and strychnia should be administered in conjunction with the remedies for the relief of the pain.

Gastro-Intestinal Disturbances.—As venous engorgement increases, the portal circulation is necessarily involved, and in consequence the liver, stomach and whole digestive tract will develop symptoms of disorder. Nausea, vomiting and fermentation of food causing gaseous distention will add greatly to the patient's suffering. For the gaseous distention, lycopodium, carbo vegetabilis, berberis, mercurius, and nux vomica, also creasote, salol, sulphocarbolate of soda, naphthalin and remedies of this class, will be of service. In nausea and vomiting relief may be afforded by small doses of calomel or ipecac. A combination in the proportion of a tenth of a grain each with one grain of bicarbonate of soda will sometimes prove effective.

As venous obstruction increases the liver becomes enlarged, congested and sometimes even pulsates, and at the same time the overdistended right side of the heart constantly receives more blood from the liver than the right ventricle can transmit through the lungs. In this condition the judicious use of purgatives will be indicated. A watery diarrhœa which is often present is an indication rather than a contraindication, inasmuch as it is a result of the congestion of the portal circulation and will be relieved by the removal of venous stasis. The best results are obtained by the use of calomel in doses of one-fourth of a grain every hour until action is obtained. Under these conditions digitalis is often worse than useless, but after the action of the purgative it may be given with good effect.

Gastric catarrh may be relieved by nux vomica, bismuth subgallate or subnitrate, ipecac, argentum nitricum, or antimonium crudum. When the secretion of mucus is abundant, and the pa strength and general condition permits, lavage will prove of sen a saline purgative will also afford great relief.

DYSPNŒA, ORTHOPNŒA AND CYANOSIS.—Absolute rest, a bo ful supply of fresh air and cardiac stimulants are the general in tions; these conditions, however, being dependent upon a varie causes, the treatment will vary accordingly. When due to ve stasis and engorgement of the pulmonary vessels the cardiac re dies mentioned and the use of cardiac stimulants, such as alc nitroglycerin, ammonia carbonate, caffein and camphor, will be ca for. When speedy action is desired remedies should be adm tered hypodermically. When dyspnœa is associated with or gravated by bronchitis, antimonium tartaricum, scilla, phosph ipecac. and stannum should be considered. Inhalations of oxy will afford relief in dyspnœa arising from this as well as ot causes. Rubbing the chest with hot sweet-oil is a simple rem which is effective and will be found useful in the more acute for of bronchial inflammation. A few ounces of normal saline solu injected subcutaneously into the abdominal wall has afforded rel especially in cases of long standing. Cold compresses applied the chest will prove satisfactory in some instances. Other reme which will sometimes prove palliative are arsenic, lobelia, cupr aceticum and hydrocyanic acid.

The cyanosis and breathlessness which arise from the prese of pulmonary œdema is the most distressing form of dyspnœa wh will be encountered, and the paroxysms of cardiac asthma wh occur may prove to be the termination of a long period of suffer This condition calls for the stronger stimulants, viz., brandy, caff and oxygen inhalations, and strychnine hypodermically.

Some attacks of dyspnœa coming on in paroxysms are due disease of the coronary arteries; here amyl nitrite and nitroglyce will be indicated.

When other measures fail, as they frequently do as the diseas progresses, and the patient's suffering becomes more intense, re source must be had to opium, which as a palliative will be pos tively essential in many cases. Morphine, hypodermically, is as suredly the best form in which to administer the remedy and i many instances it will have an excellent effect.

Dyspnœa dependent upon hydrothorax should be relieved at once by aspiration ; in some cases counter-irritation in the form of blisters will answer.

PALPITATION.—The recognition of the cause is essential in order to meet this symptom. If it arises from excessive hypertrophy as evidenced by a full, tense radial pulse, attended with a sensation of constant throbbing in the præcordium, aconite, belladonna, cactus grandiflorus, veratrum viride will be indicated. When palpitation is induced by nervous influences, ignatia, moschus, cactus grandiflorus, arsenicum, the bromides, Hoffmann's anodyne well diluted, or alcoholic stimulants in small quantities act well. The ice-bag or Leiter's coil applied over the præcordium will relieve in many instances.

Attacks of tachycardia may be controlled by the same remedies, notably ignatia, cold applications and Hoffmann's anodyne in doses of fifteen to thirty drops.

NERVOUS PHENOMENA.—In many instances, especially in the later periods of the disease, the patient will evince the most extreme restlessness, irritability and insomnia. Sleep when it does come may be disturbed by distressing dreams or attacks of palpitation. For the restlessness and irritability, ignatia, arsenicum, sodium bromide or potassium bromide may be used. For insomnia, belladonna, passiflora incarnata, the bromides, codein, hot alcoholic drinks at bed-time, sulphonal and trional. In some instances morphine will be found to be the only remedy that will afford relief.

DROPSY.—This symptom is one of the most serious significance and at the same time the most troublesome. Rest, careful diet and attention to the state of the bowels are most essential, and in the milder forms will often prove sufficient, but in the severer grades more active measures will be demanded.

The patient should be placed in such a position as to favor the return flow of the circulation. Gentle massage should also be practiced. While it is desirable to restrict the amount of fluids ingested, it is manifestly often impossible to give solid food on account of the condition of the stomach, hence, while the food must be fluid, the quantity, however, should be limited. In severe cases about two ounces every two hours will suffice. It is important that the bowels should be kept open, but free purgation must be avoided.

Digitalis is of great service, especially in the form of the i
one-drachm doses of which should be given every two
hours, or it may be administered in alternation with son
remedy.

Apocynum Cannabinum is a diuretic of great utility; it wil
the urinary secretion rapidly. Ruddock recommended it
nation with cactus. When a fresh infusion can be obt
should be preferred, but generally this is impossible and t
ture must be employed. The objection to the drug is its
to produce nausea.

Scilla is an important remedy in cardiac dropsy, but
used with caution when the kidneys are involved. It may
with digitalis alternately.

Caffein, on account of its decided diuretic action, may
quently advantageously administered as a substitute for
when it is desired to take advantage of its less irritating qu
the gastric mucous membrane and of its greater rapidity o
It may be given as previously mentioned.

Apis is useful and will often afford great relief when its
indications are present. It is more especially indicated w
phritis is present as a complication.

Arsenicum finds its sphere in those forms of dropsy w
marked by œdema of the face, hands and feet, by great
thirst, red tongue, dyspnœa and emaciation. It is also o
in the aged.

Potassium acetas or bitartras are useful diuretics. They are heavy and distended, and the skin threatens to rupture, mechanical measures for relief must be employed. Simple cutaneous incisions, the use of Southey's fine silver canula or trocar are the methods employed. Whatever plan is selected careful antisepsis should be observed; the skin should in all cases be cleansed with ether, alcohol and sublimate. When a trocar is used it should be inserted obliquely into the œdematous tissue and the stylet then withdrawn. The surface of the skin surrounding the point of insertion should be dusted with some antiseptic powder. A rubber tube may be attached to the canula to conduct the fluid over the side of the bed. The tube and canula should be fixed in position by adhesive strips to prevent traction. One or two tubes in each leg are usually sufficient. When the canula becomes plugged insert the stylet and remove the obstruction or insert the canula again. When incisions are decided upon four should be made, two on each leg, one each side of the malleolus. The incisions should be long and sufficiently deep to reach the subcutaneous tissue. Cover the part with aseptic gauze, then a thick layer of absorbent cotton and one of rubber, the latter should be so arranged that a part of the heel is left uncovered. As the patient sits up (at this stage of the disease the sitting posture is more or less constant) the fluid drains down through the uncovered part and is caught on cloths, or in a basin. A large amount of fluid will escape by this means and the relief will be decided.

NEPHRITIC COMPLICATIONS.—In the latter stages of the disease the urine may become scanty and albuminous and more or less filled with casts. Uræmic symptoms may appear and the danger of a fatal termination becomes imminent. Under these circumstances non-nitrogenous liquid food, confinement to bed and the administration of remedies directed to increase the action of the skin and kidneys, and purgatives will be indicated. Nitroglycerin often acts well, one to two drops of the first centesimal dilution may be given every two to four hours.

Special Valvular Lesions.

The influences which enter into the ætiology of chronic heart disease, the pathology, clinical course and treatment as previously discussed, necessarily all apply to the various forms of valvular deficiencies. Nevertheless, since special lesions are more prone to arise in connection with certain morbid influences, and manifest pathological changes and symptoms more or less characteristic, the several forms of valvular defects must be considered individually.

Mitral Insufficiency.

Mitral insufficiency is incomplete closure of the mitral valve, in which during ventricular systole there is flow of blood backward into the auricle, all of which should have been propelled into the aorta.

Ætiology.—The causation of mitral insufficiency is varied. The lesion may result from disease of the valve segments and chordæ tendineæ or from the effects of morbid processes in the myocardium. The most frequent cause is rheumatic endocarditis. Endocardial inflammations of other types may likewise, though less frequently, produce the same results. Under these influences the valvular segments and orifice are the usual seats of the disease process. The chordæ tendineæ and columnæ carnæ may also become affected, with the result that the valve cusps will not be able to adapt themselves to the opening and regurgitation is the consequence. The lesion is also common in connection with arterio-sclerosis, in which the heart may be the principal seat or only a part of the degenerative process. In such cases the atheromatous process is frequently found to have involved the mitral area by extension from the aortic.

The changes in the myocardium which are operative in causing mitral incompetence are those which result in dilatation of the left ventricle, with enlargement of valvular orifice and consequent failure of the mitral valve to close completely. The chief causes of this group are dilatation arising secondarily to aortic disease on account of increased blood-pressure, grave anæmia and acute febrile diseases, and degenerations, especially those associated with advancing years. In the first instance the change may be of a mild character; in the second they are more or less amenable to treatment, while in the last they are always grave.

Conditions of hypertrophy which compensate a valvular defect are liable in time to fail and become the source of mitral incompetence. The heart muscle, from the long continued, abnormal blood-pressure, will weaken and dilate, and in this way enlarge the valvular orifice, and thus the valves will no longer be able to close effectively and relative insufficiency follows. This condition is common in the right side of the heart, but in the later stages of hypertrophy it is also frequent in the left, where it occurs in consequence of increased blood-pressure, Bright's disease and arteriosclerosis.

Mitral insufficiency may appear in connection with chorea. In a majority of patients suffering from this affection, some time during the course of the disease, or throughout its entire course, the presence of a systolic murmur will be noted. The rheumatic origin of such cases is often evinced by positive signs.

Finally, mitral insufficiency may be caused by neoplasms.

Morbid Anatomy.—In those forms of mitral incompetence which arise from endocarditis, the changes at first may not be extensive, but in time the cusps will undergo marked thickening and contraction attended with deposits on their surface, especially on the ventricular side.

The papillary muscles will thicken and the chordæ tendineæ will either lose their tone or become shortened or adherent to the walls of the ventricle and bind down the valves. More or less constriction of the orifice is common in mitral insufficiency arising from endocardial inflammation, hence stenosis is often present with mitral regurgitation. Fusion and adhesion of the cusp margin may also occur and give rise to obstruction.

In rare instances a valve segment may be ruptured as the result of strain, aneurysm or ulceration, or it may be detached from its insertion at the base.

Degeneration will likewise prevent the valve from acting normally by producing rigidity and contraction and often distortion of the cusps. In addition there will be atheromatous deposits, fatty, fibrous or calcareous. In some instances the leaflets are but slightly involved, yet have vegetations or concretions on their surfaces which interfere to a material degree with the function of the valve. In cases of long standing there may be a ring of calcareous or fibrous deposit around the mitral orifice.

When incompetence arises from changes in the myocardium the pathological conditions are somewhat varied. Enlargement of the auriculo-ventricular opening may arise from loss of tone of the muscular fibres encircling it, as the result of inflammation of these structures, so that the cusps will be unable to adapt themselves effectively to the widened orifice. Again, the ventricle may undergo enlargement to a greater degree than the papillary muscles and thus lead to imperfect valvular action.

The auriculo-ventricular openings, it must be remembered, during diastole are ovoid or circular in shape, but during systole, owing to the contractile influence of the muscles at the base of the heart, they become buttonhole-like slits. It is, therefore, not difficult to see that any impairment of the heart muscle, such as relaxed and paretic conditions, effects of acute inflammations, cicatricial contractions from chronic endocarditis, or degenerative change in the heart walls may readily interfere with the proper closure of the valves and lead to incompetence. It must be borne in mind that these causes and effects are not by any means always clearly separable, contractions and adhesions of the cusps, vegetations and atheroma will be found in association with enlargement of the orifices and degenerations of the myocardium.

In the advanced conditions which follow failure of the mitral valve obstruction of the circulation and consequent perverted nutrition will produce grave anatomical changes in distant organs. The lungs will be engorged with dark blood and abnormally dense from proliferation and fibrous tissue. The lung tissue itself becomes toughened, the capillaries dilated, with the walls thickened and varicosed. In long continued and moderate passive hyperæmia the overgrowth of connective tissue is attended with pigmentation of the tissues from breaking down of the blood corpsucles, resulting in what is known as brown induration. In many cases the lesions of the lungs will present various types. Effusions into the pleural cavity may arise and produce collapse of certain portions of the lung. Pulmonary infarction may occur in various localities and at various periods in the course or the disease. The sites of the old infarctions will be recognized by the presence of the pigmented indurations on the lung surface, accompanied in some instances by a depression on the pleural surface. In recent cases the corresponding area of the pleural surface may be covered with a yellowish pleuritic inflammatory patch.

The liver will be enlarged, the intralobular capillaries dilated and their walls thickened. Dark-brownish stellate spots mark the centre of each lobule on a yellowish ground formed by the bile-stained liver cells. Clusters of dilated venous capillaries in the centre of each may encroach and cause atrophy or fatty degeneration of the latter. Mixed with these points of discoloration there is a general reddish hue which arises from disorganized blood. The internal structure on section discloses a characteristic mottled appearance known as "nutmeg liver;" a condition generally regarded as pathognomonic of mitral disease.

The stomach shows dilatation of the veins with congestion of the mucous membrane and the presence of tough or fluid mucus in abundance. The spleen is enlarged with intense venous engorgement. The kidneys are abnormally firm from cyanotic induration. The pyramids are engorged. Blood may flow from the glomeruli into the tubules. Infarctions are common. In recent cases they appear as wedge-shaped bodies with their base toward the cortex and apex to the hilum. In old cases a deep depression on the surface with cicatricial tissue may indicate the presence of old emboli.

The membranes of the brain also shows evidence of venous engorgement.

MECHANICAL EFFECTS OF THE LESION.—The normal action of the mitral valve does not permit of any reflux of blood. On the other hand, when the segments, for any reason, fail to effect perfect closure, a portion of the blood flows back into the auricle whence it came. This current meets that from the pulmonary veins coming into the left auricle, and causes obstruction and disturbance of the normal flow. There are, therefore, two streams flowing simultaneously into the left auricle during systole of the ventricle. The effect of this two-fold flow is over-filling and ultimate dilatation of the auricle, at the same time it increases the work put upon it. The consequences are some compensatory hypertrophy of its walls, but the latter, being weak and incapable of much hypertrophy, remain enfeebled. During the next diastole the contents of the distended auricle are propelled forward under increased pressure into the left ventricle. This abnormal quantity of blood in turn produces over-distention of that chamber. The ventricle now, in discharging its contents into the aorta is unable to do so completely, and some of from the right auricle to the right ventricle becomes impeded. Dilatation of the right auricle follows from increased pressure, with the result of general venous congestion. When the right side of the heart loses power and fails, a smaller amount of blood is supplied to the left ventricle and, in consequence, the quantity flowing into the aorta is lessened. For this reason the arterial tension is lowered in the later periods of the disease.

A summary of the mechanical effects of mitral insufficiency is as follows: Regurgitation of blood from left ventricle into left auricle, distention and dilatation of left auricle; backward pressure in pulmonary vessels in consequence of the flow from right venticle being impeded; the result of this pressure is dilatation and hypertrophy of right ventricle. Dilatation and hypertrophy of the left venticle also occurs in consequence of the extra amount of work put upon it.

Symptoms.—Manifestations of symptoms in mitral insufficiency depend largely upon the rapidity with which the pathological changes progress. If slow, the heart will have time to adapt itself to the changed conditions, and either prevent or postpone the development of symptoms. On the other hand, if the changes are active and quickly result in damage to the heart structure, symptoms will develop in a correspondingly brief period.

In those cases where the favorable condition of the patient and slowness of development allow compensation to be effectively sustained for an indefinite period, there is often complete absence of symptoms. Disturbances, on the other hand, may appear, but they are mild and transitory. Shortness of breath on slight exertion or from nervous influences is often the first and most prominent symptom and, in many instances, the only one observed for a considerable time. Palpitation is also common, being most frequent in young and nervous subjects.

When the compensatory power has broken down, symptoms of a mild and somewhat temporary character which may have been present will be displaced by those of persistently increasing gravity. Failure of compensation means inability of the right ventricle to propel the normal amount of blood through the left side of the heart. The natural sequence is engorgement of the venous system which originates in the right side of the heart and extends toward the periphery implicating the viscera, mucous membranes and extremities.

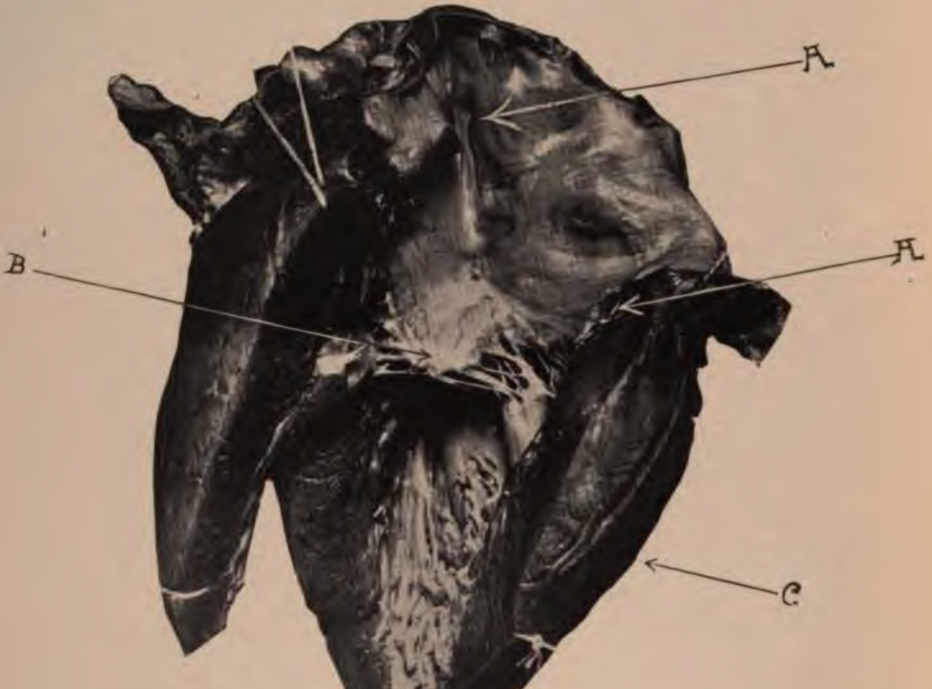


FIG. 19.—Mitral insufficiency with thickened and indurated valve the result of benign endocarditis. A, valvular orifice laid open; B, thickened valve; C, wall of ventricle showing hypertrophy.

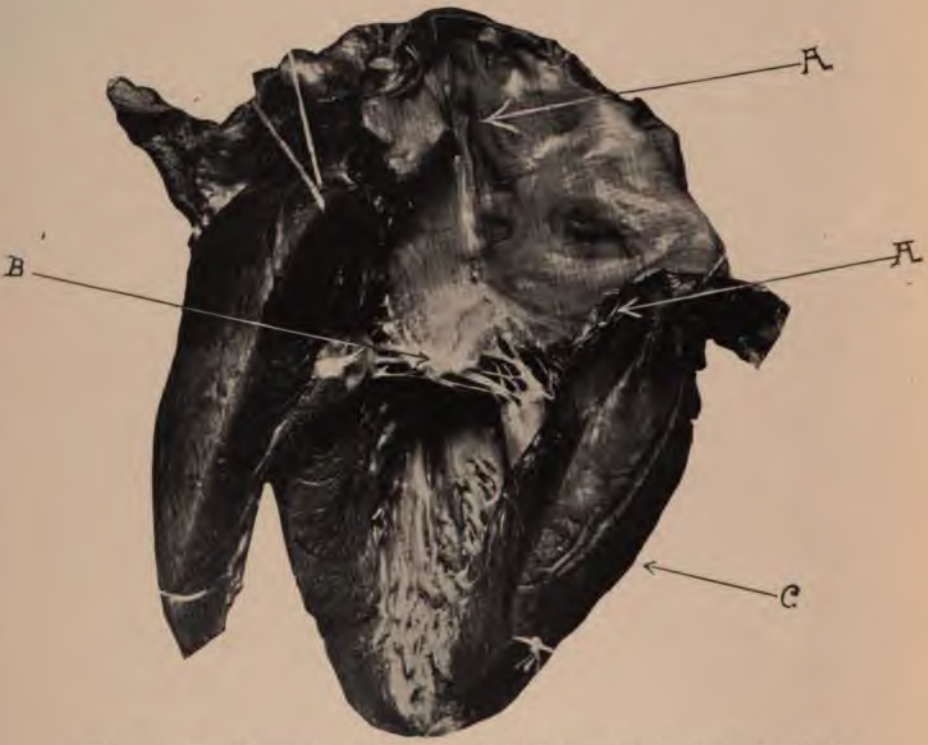
A sense of distress or weight in the region of the præcordium is a common symptom. Sometimes there is pain. Palpitation is frequent. These sensations will, for the most part, be experienced on slight exertion. Evidence of disturbance of the cardio-pulmonary circulation are decided. Breathlessness is easily induced from trivial causes; cough, sometimes attended with watery blood-streaked expectoration, will be troublesome and often persistent. Sometimes there is hæmoptysis. Sooner or later the long train of secondary symptoms which have already been described manifest themselves. There is gastro-intestinal irritation and catarrh, as indicated by nausea, vomiting and intestinal disturbances, enlargement of the liver and spleen, and grave nephritic complications, as evidenced by the passage of scanty urine containing albumin and casts. Dropsy soon follows, appearing first in the feet and ankles and extending rapidly upwards.

The countenance often shows that the circulation is deficient; the cheeks have a dusky appearance, the lips and nostrils are cyanotic and the superficial capillaries congested and sometimes arborescent. Jaundice of a mild degree is quite common and when associated with cyanosis produces a peculiar greenish color. Clubbing of the fingers may also be observed.

The pulse in mitral insufficiency when compensation is sustained suffers little or no material change except some lowering of pressure. Sphygmographic tracings present an almost natural appearance. On the other hand, when compensation is broken, the pulse is feeble, compressible, irregular and rapid.

Cerebral symptoms are frequent. Giddiness, faintness and sensation of fullness in the head may be experienced. Insomnia is also common and is especially liable to appear in this form of valvular deficiency. In advanced conditions, delirium, illusions and hallucinations may manifest themselves.

PHYSICAL SIGNS.—*Inspection* may disclose some prominence of the præcordial region, in the second and third interspace to the left of the sternum, especially in children. The apex-beat may be to the left of the normal position, and in some instances a little lower. It may appear on a line with the nipple or beyond it. Its area of diffusion may be increased, extending to the left and downward, corresponding with the hypertrophy of the left ventricle. On account of



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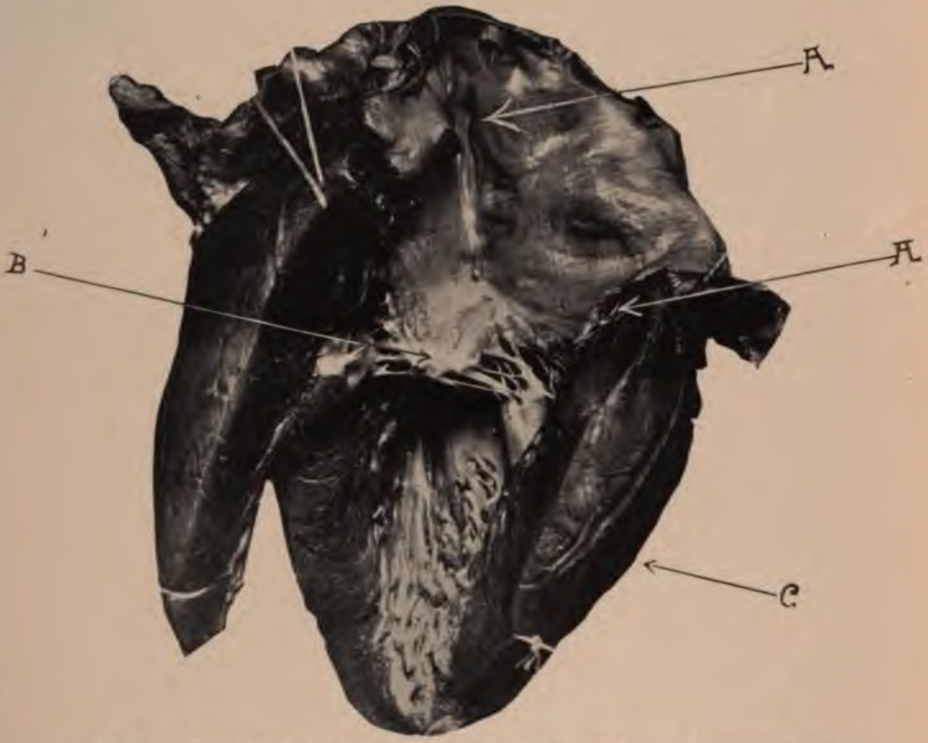

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its diffuse character it is sometimes difficult to determine the situation. Epigastric pulsation is common, especially in connection with dilatation of the right ventricle. There is sometimes a distinct movement from above downward in the third, fourth and fifth intercostal spaces, and a heaving in the epigastrium. There may be also a pulsation in the second left intercostal space. When failure of the right side of the heart occurs pulsation of the cervical veins and sometimes slight jaundice may be observed.

Palpation shows variation of the apex-beat. When compensation is attained it may be more forceful than normal. When compensation has given way the apical impulse grows more feeble and diffuse, and as the inadequacy advances, irregular and arrhythmic. Sometimes a sharp impulse accompanying the second sound, caused by pressure within the pulmonic artery, may also be felt. In the earlier stages the radial pulse suffers little or no change except that it may be lowered at times; later it becomes frequent, compressible and irregular. It always has a direct relation to the apex impulse.

Percussion reveals enlargement of the normal area of dulness. The increase is to the left, extending frequently to the axillary line, and to the right from one-half to one inch beyond the right sternal line. The tendency of dilatation and hypertrophy is to cause a widening of the area of dulness rather than vertical enlargement.

Auscultation naturally affords the most important evidences. A systolic murmur is heard in the mitral area, transmitted to the left toward the axilla and posteriorly under the left scapula. The point of maximum intensity is at the apex.

The murmur varies in intensity and extent; it may either be a simple accompaniment of the first sound or it may entirely displace it. The conduction of the sound is important and also, though rarely, subject to variations. Sometimes it is heard with an intensity at the base of the heart to the left of the sternum as great or nearly equal that observed at the apex. This has been explained as due to the transmission of the murmur to the left of the auricle through the auricular appendix which surrounds the pulmonary artery and lies in close contact with the thorax. In some instances the murmur may be heard over the entire chest wall.

The character of the murmur is usually soft, sometimes, though rarely, terminating in a musical tone. Occasionally it is rough. The second pulmonic sound of the heart is intensified over the second intercostal space and often at the apex, due, as mentioned, to increased tension of the pulmonary vessel from hypertrophy of the right ventricle. When the tricuspid valves become relatively inadequate from dilatation of the right ventricle, this accentuation of the second sound disappears.

Combinations of murmurs may be heard, and sometimes a rough presystolic sound. When secondary dilatation of the right ventricle occurs, giving rise to relative insufficiency, its characteristic murmur may be heard at the ensiform cartilage.

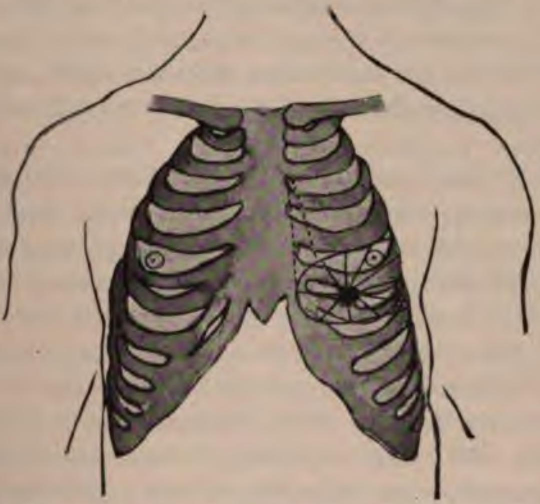

FIG. 20.—Area of the mitral systolic murmur, the arrow showing the usual direction of transmission. The black circle indicates the usual locality of maximum intensity. The dotted lines upward along the border of the sternum show an occasional, but not uncommon, direction of transmission.

Diagnosis.—Mitral insufficiency is readily recognized by the presence of a systolic murmur, heard with its maximum intensity at the apex, transmitted to the left and audible laterally and posteriorly beneath the left scapula, by the increase of the area of dulness, and by the accentuation of the pulmonic valve sound.

Although the diagnosis of mitral insufficiency is not usually attended with difficulty, there are certain other conditions accomits diffuse character it is sometimes difficult to determine the situation. Epigastric pulsation is common, especially in connection with dilatation of the right ventricle. There is sometimes a distinct movement from above downward in the third, fourth and fifth intercostal spaces, and a heaving in the epigastrium. There may be also a pulsation in the second left intercostal space. When failure of the right side of the heart occurs pulsation of the cervical veins and sometimes slight jaundice may be observed.

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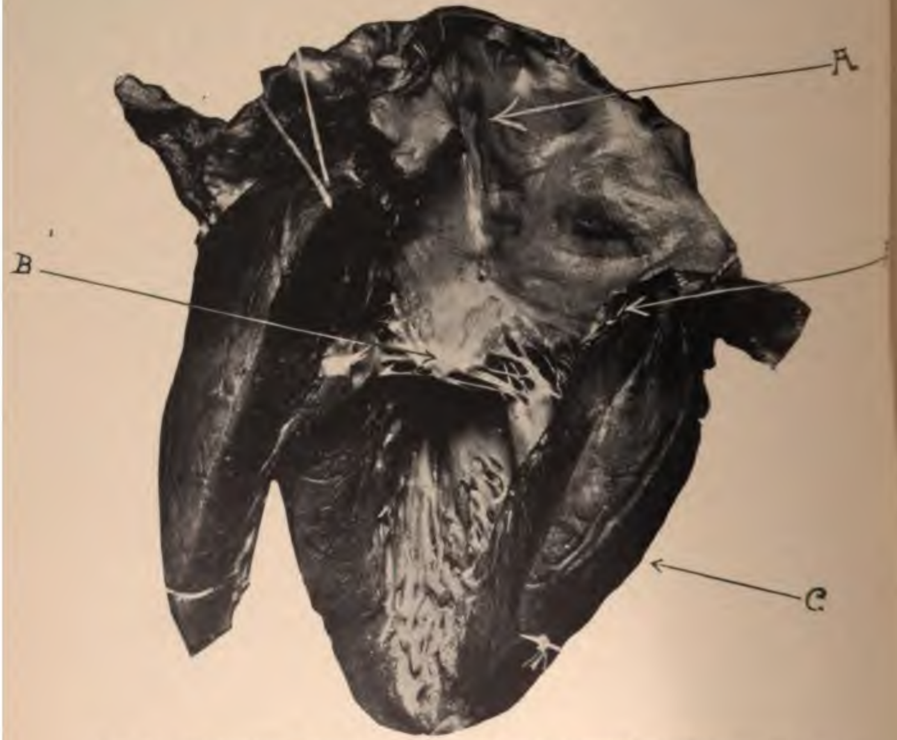

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A sense of distress or weight in the region of the præcordium is common symptom. Sometimes there is pain. Palpitation is freuent. These sensations will, for the most part, be experienced on light exertion. Evidence of disturbance of the cardio-pulmonary circulation are decided. Breathlessness is easily induced from trivial causes; cough, sometimes attended with watery blood-streaked expectoration, will be troublesome and often persistent. Sometimes there is hæmoptysis. Sooner or later the long train of secondary symptoms which have already been described manifest themselves. There is gastro-intestinal irritation and catarrh, as indicated by nausea, vomiting and intestinal disturbances, enlargement of the liver and spleen, and grave nephritic complications, as evidenced by the passage of scanty urine containing albumin and casts. Dropsy soon follows, appearing first in the feet and ankles and extending rapidly upwards.

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panied with a murmur at the apex and also associated with the first sound which may be confused with it. These are aortic stenosis, anæmic bruit, tricuspid regurgitation and pericarditis.

A mitral regurgitant murmur may be distinguished from that of aortic stenosis by the fact that the maximum intensity of the former is heard at the apex, the area of transmission is to the left and posteriorly, there is accentuation of the pulmonic sound, and enlargement of both ventricles. In contradistinction to these features, aortic stenosis has its point of maximum intensity at the base of the aorta, its bruit is transmitted upward along the great vessels of the neck, it is not accompanied by accentuation of the pulmonic sound and the left ventricle is enlarged along its entire course. Other points of difference are the harsher character of the aortic stenotic murmur, the thrill at the base of the heart, the age of the patient and the pulse.

Mitral regurgitant murmurs may be counterfeited by those of functional anæmic origin, but there are several marked features which in most instances render differentiation a comparatively easy matter. In the first place, the causal influences must be taken into consideration. In mitral disease there often is a history of rheumatism or some other condition which may give rise to endocarditis. While in functional anæmic murmurs there is a debility and marked anæmia. It is true that anæmia may also appear in association with conditions which produce endocardial changes, but the physical signs show the following differences: On inspection, it will be at once observed that functional murmurs are attended with decided pallor and blanching of the mucous membranes, while in mitral disease there is the dusky hue of the lips and cheeks, and pallor, if present, is usually not so marked. Palpation in anæmic murmur will fail to show either displacement of the apex-beat or augmentation of the impulse. The pulse tension is prolonged and the arterial pressure increased. In mitral insufficiency the converse of these conditions will be observed; namely, increase of impulse, displacement of the apex and lowering of the pulse tension. In anæmic conditions dilatation is not marked. In about one-half of all cases there is dilatation of the right auricle causing dulness about or to the right of the sternum.

The differences on auscultation are important. Mitral insufficiency has its characteristic transmission to the left, as mentioned; while the anæmic murmur, although it may be heard synchronously with the first sound of the heart with its greatest intensity at the apex, does not possess any special area of transmission, but is generally diffused in all directions. There is also absence of accentuation of the second pulmonic sound. It is, furthermore, often accompanied by a general bruit more or less audible over the whole præcordium. There may also be venous pulsation in the vessels of the neck.

Tricuspid insufficiency (*vide*), having its characteristic seat, maximum intensity and area of transmission materially different from those of mitral insufficiency, may usually be clearly distinguished. The presence of pulsations in the cervical veins is conclusive.

Pericardial friction murmurs, while audible with greatest intensity at the base of the heart, differ from the sounds of mitral regurgitation in that they do not accurately correspond with the heart-sound and that they vary as the position of the patient is changed. Neither of these characteristics belong to mitral regurgitation. The history of the case and ætiology are also important factors.

It is difficult and often impossible to determine whether a condition of mitral insufficiency arises from changes in the valve cusps or is dependent upon dilatation of the ventricle (relative insufficiency). The distinction rests mainly upon the character of the murmur and the history of the case. Murmurs resulting from lesions of the cusps are said to be generally harsher and louder than those due to dilatation, but the contrary may also be observed as the writer has verified by post-mortem examination. Again, lesions of the cusps are more liable to occur in young subjects who have suffered from endocardial inflammation, particularly that of rheumatic origin; while relative insufficiency is more frequent in middle life in persons whose history points to chronic renal disease, gout, arteriosclerosis, alcoholism or syphilis. Furthermore, when mitral insufficiency exists in connection with chronic kidney disease, with high arterial tension and hypertrophy of the left ventricle, it may be presumed that the condition is one of relative insufficiency.

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Prognosis.—In mitral insufficiency the outlook is more favorable than in any other form of valvular defect. The numerous factors which directly and indirectly influence the course of the disease, and the many circumstances and conditions which bear u its future and which render the question difficult, have been d- cussed under the general subject. The fact that in many pers the lesion exists for years without exciting apprehension or giv evidence of disturbance has been mentioned. This is particula true in children in whom compensatory hypertrophy is often f established and effectively sustained.

The prognosis will, in a great measure, depend upon the rapid with which the pathological processes develop. As a rule, it is sl and insidious, and the heart is thus enabled to adjust itself to t new conditions and thereby to establish compensation. In those i stances where the lesion develops suddenly and the destructive i fluences operate rapidly, there can be little or no compensation a the symptoms are severe and the prognosis correspondingly grav Such conditions may occur as the result of severe forms of acut endocardial inflammation where the damage of the valve structure has been excessive, and in case of rupture of the valve. The latter although attended with alarming symptoms, intense dyspnœa, se- vere præcordial pain and symptoms of general collapse, may allo the patient to rally and live from a week to several months or eve a year and more.

Furthermore, the prognosis will be influenced by accompanying complications, particularly mitral stenosis and disease of the hear walls. Again, the presence of acute diseases, especially those o the lungs, is unfavorable.

In a large number of cases of mitral insufficiency care and treat- ment will accomplish beneficial results, and many persons with this form of lesion live to old age. In the earlier stages of weakening compensation therapeutic measures often prove successful in re-es- tablishing the equilibrium of circulation or, at least, in averting for a time its final failure.

Treatment. The methods of treatment previously discussed are generally applicable. When the lesion appears in the course of acute febrile affections, as it frequently does, it will be possible to observe its development. Under such circumstances every effort should be put forth to restrain the increasing inroads of the disease. [illegible] by complete rest and the admin- [illegible] remedy result is constriction of the orifice. In some cases the adherent valves are turned down into the ventricle, producing a hollow cone or funnel-shaped condition. In others the contraction appear as a narrow slit, generally termed a button-hole constriction. The former is more common among children, while the latter belongs to adult life or advanced cases. Associated with these changes there is fibrous thickening in the adjacent tissues, and, in the case of the button-hole constriction, calcareous infiltration with plates having the hardness of bone. The chordæ tendineæ are thickened and shortened and the musculi papillares dense.

Another variety of pathological process is that in which the surface of the valve is the seat of deposits. Verrucose vegetations and granulations or tough fibrinous masses may develop on the valve and invade its substance. These bodies protrude and obstruct the flow of blood. Their usual site is on the auricular surface of the valves a little distance from the border. Similar changes in the chordæ tendineæ which produce rigidity in the cusps may also be observed.

Still another type of change is that of atheroma and sclerosis of the cusps. There is thickening with atheromatous and calcareous deposits. The result is rigidity and deformities of the valves, which not only interfere with their function, but also cause them to act as obstructions to the blood-current.

The tendency of chronic endocardial inflammation to progress in the instance of mitral stenosis is an important feature in the pathological chances.

The degree of contraction which the mitral orifice undergoes may sometimes be extreme. In some instances it has been so great that a pencil could scarcely be passed through the opening. Excessive contraction, however, has been shown to be compatible with life. It has been stated that compensation is impossible only when the orifice has become reduced to one-tenth of its normal size. The size of the opening in health has been roughly estimated as capable of permitting the insertion of the index and middle fingers, but post-mortem rigidity may so contract the ring as to render this estimate unreliable.

MECHANICAL EFFECTS OF THE LESION.—The changes in the heart which characterize mitral stenosis are principally manifested tics of Edinburgh* the average age of those presenting themselves for treatment ranged between 20 and 30 years.

Sex appears to influence the causation, as the condition is more prevalent among females than males. This is owing to the fact that the affections which are ætiologically related to it are more prone to occur in the former sex.

The diseases which result in obstructive processes of the mitral valve are the same as those which produce insufficiency. The ætiology thus depends chiefly upon endocarditis; that arising from rheumatism being by far the most frequent. In a certain proportion of cases it will be impossible to ascertain, even after the most careful inquiry, any definite origin, as there will be complete absence of any affection which could be regarded as its source. In some instances, however, it may happen that years after the first manifestation of the lesion irregular types of rheumatism appear which will point to that affection as the latent cause.

Rare causes of mitral obstruction are protruding aneurysms of the ventricular wall, or of the valve, and neoplasms. Cases of large exuberant growths on the aortic valves extending down into the mitral orifice and causing obstruction have been recorded.

Morbid Anatomy.—The changes at the valvular orifice present a wide range of differences, varying from a simple roughened surface, without appreciable constriction, to a high degree of obstruction from contraction and deformities of the valve structures.

The seats of lesion are the auriculo-ventricular ring and the cusps and structures connected with them. In the former the results of endocarditis are apparent and vegetations and sclerotic degenerations may be observed. In young subjects vegetations predominate, while in cases of long standing both vegetations and sclerosis are common in association with calcification.

The changes connected with the valve structures and chordæ tendineæ are of more frequent occurrence. Here the lesion presents several types or groups of manifestations. In the first instance the margins of the cusps become joined by fibrous adhesions, and, later, the two curtains show complete fusion. The gree, the right auricle is also dilated, and in advanced cases tricuspid insufficiency follows.

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* Diseases of the Heart and Aorta, G. A. Gibson, M.D., Edinburgh, 1898.

The chain of consequences of mitral obstruction may be briefly summarized as follows: Obstruction of mitral valve, abnormal tension in the left auricle, auricular dilatation and hypertrophy, obstruction to the blood-flow from the pulmonary veins, increased work of the right ventricle, dilatation and hypertrophy of right ventricle.

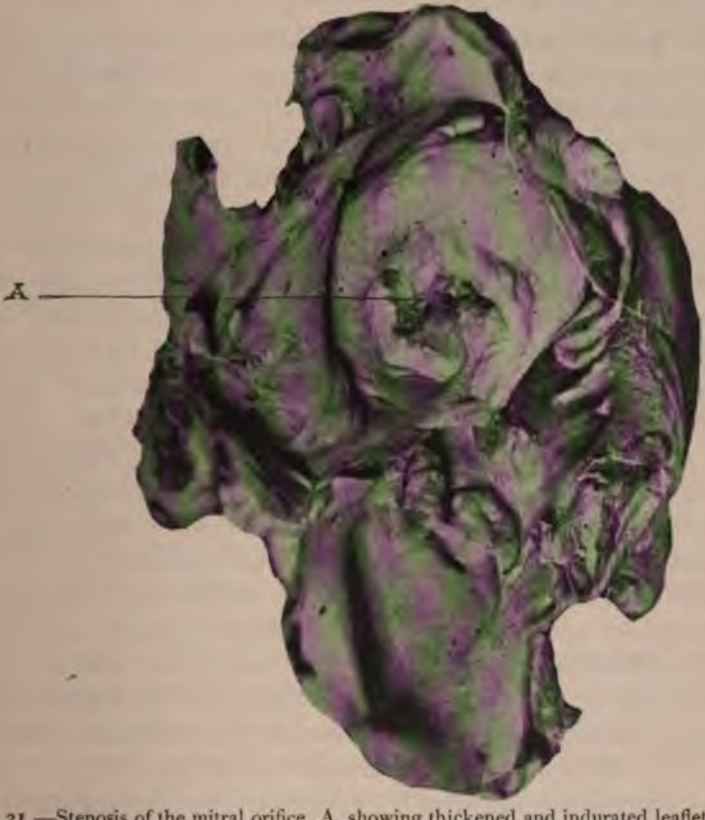

FIG. 21.—Stenosis of the mitral orifice, A, showing thickened and indurated leaflets of the valve and button-hole opening. View from left auricle into the mitral orifice.

The secondary changes in the general circulation and distant organs, which follow failure of compensation in mitral stenosis, are similar to those which result from mitral insufficiency. Brown induration of the lungs and dilatation of the pulmonary capillaries in the left auricle, the walls of which undergo more or less thickening, and in the right ventricle which becomes somewhat dilated and hypertrophied. The obstruction first makes itself felt in the auricle. When the equilibrium of circulation is sustained there may be simple hypertrophy, the extent of which will depend upon the amount of extra work which the auricle is called upon to perform. But when the heart begins to fail, dilatation accompanies hypertrophy and in the last stages the walls of the auricle may become very thin. Frequently its cavity will be found to contain ante- and post-mortem blood clots.

The effect upon the left ventricle is varied. The influence to which it is at first subjected is directly opposite to that exerted upon the auricle, namely, a diminution of blood-supply, the consequence of retardation of the flow from the auricle. The result is that there is no hypertrophy, and inasmuch as less work is put upon its muscle there may be, in some instances, some diminution in size. This applies only to uncomplicated cases, with complete compensation. On the other hand, it will undergo hypertrophy as soon as failure of compensation makes itself felt, for the reason that backward pressure, which thereupon results, exerts its influence back upon the venous system, so that the flow of the arterioles into the latter is obstructed; this leads to augmentation of the work which the left ventricle is called upon to perform and hypertrophy ensues. Another explanation is that of interference with the action of diastolic aspiration of the left ventricle which must necessarily result from the presence of mitral obstruction. If the diastole be regarded, as it is by many authors, as an active muscular process, it follows that a certain degree of hypertrophy will result by interference with this function. The increased power of the heart which follows as the combined result of hypertrophy of the left auricle, and the increased pressure of the circulation from the stenosis, for a time may remain evenly balanced. Usually at a comparatively early period the auricle becomes unable to sustain this power and compensation is no longer sustained. Then follows retardation of the blood-flow of the pulmonary veins into the auricle and the vascular tension in the lungs and right ventricle becomes greater. In conseque the right ventricle in order to overcome this pressure beco markedly dilated and its walls hypertrophied to an excessive some of its tone. Broncho-pneumonia may also occur, and when the impediment to the circulation is more serious pulmonary œdema may develop. In some instances the continuous high pressure to which the pulmonary vessels are subjected may result in chronic bronchial catarrh and sclerosis and atheromatous degenerations of the walls of the bronchial vessels.

Pain cannot be said to characterize mitral obstruction, though it may be present. Its favorite seat is the anterior surface of the chest, left shoulder and arm.

When the disease has advanced sufficiently to affect the right side of the heart, and the latter fails, tricuspid regurgitation and its attendant symptoms will develop. The patient suffers from gastro-intestinal catarrh ; there is gastric disturbances, derangements of the bowels and a sense of weight in the region of the liver, which organ may undergo an excessive degree of hypertrophy, sometimes displaying pulsations.

Dropsy arises, and the evidences of hepatic congestion and obstruction of the portal circulation are seen in ascites, which may make its appearance before œdema of the lower extremities. General anasarca may also follow, but it is not so frequent with mitral stenosis as it is with insufficiency.

During the course of the disease intercurrent febrile attacks may arise from exacerbations of endocardial inflammation, which greatly exaggerate the condition. Cerebral symptoms are very common in the form of headache and insomnia.

The formation of emboli is another feature. Apart from inflammatory and ulcerative processes emboli occur more frequently with this than any other form of valvular disease. They are due to fibrinous coagula which form in the musculi pectinati of the left auricle or appendix, or columnæ carneæ of the ventricle, and which become detached and are carried into the circulation.

There is nothing in the countenance of the patient suffering from mitral stenosis which can be called characteristic. In some instances, up to an advanced stage, there may be neither pallor, a dusky hue nor anxiety, but, on the contrary, a general healthful appearance. On the other hand, even in cases with a slight degree of obstruction there may be a tendency to a dusky flush high up on the cheeks, which some writers consider as eminently characteristic.

are frequent. Hydrothorax is liable to occur when the right of the heart fails.

Symptoms.—The lesion may be present without giving evidence of its presence other than its physical signs, althou often, even when compensation remains complete, breathlessness some unusual exertion will be sufficiently marked to attract atte tion.

The general symptoms, in comparison with those arising fro other valvular affections, are characterized by a greater prominenc of conditions dependent upon pulmonary stasis, by the presence excessive enlargement of the liver, by the greater frequency of cites, the less frequent appearance of general anasarca, and by th more marked tendency to the formation of emboli. As soon compensation becomes feeble, disturbances of the pulmonary circu lation appear and evidences of stasis and defective aëration of th blood are speedily observed. A feature of the pulmonary hype æmia of mitral obstruction is that it may not be constant as th auricle may, at times, have an opportunity to empty itself. Breathlessness is one of the first appearing symptoms and at the sam time one of the most persistent. It may arise from slight effort emotional influence. In some instances it is the cause of little mor than temporary annoyance, while in others, when compensation decidedly defective, it is the source of great distress, being more less constant and greatly aggravated by any effort. Paroxysms dyspnœa are common, and may be regarded as cardiac asthma.

In conjunction with the shortness of breath there may be cough which may at times be dry and hacking, or accompanied by watery expectoration which is occasionally blood-streaked. Hæmoptysis may occur from rupture of a pulmonary vessel.

As a further consequence of pulmonary circulatory disturbances there is a very decided tendency to bronchial inflammation ; the patient will be very susceptible to atmospheric changes and will take cold on the slightest provocation. The resulting bronchitis ofte proves deep seated and obstinate and may aggravate any tendency to weakening compensation. In such cases, even when the respiration has been previously free from discomfort, it may in intense dyspnœa with asthmatic breathing. This distressing sy tom may disappear as the bronchitis improves and the heart rec num in the fourth and fifth interspace. The impulse of the hypertrophied right ventricle may be felt in the epigastrium.

In advanced cases the apex may impart sharp, abrupt impacts like tapping, at the same time unlike the full, forcible impulse of hypertrophy.

By far the more important feature is the presence of a presystolic thrill at the apex or just internal to it; this, however, is not always appreciable. It may be felt to the best advantage over the fourth and fifth interspaces within the mammary line, and during inspiration. It begins during diastole, that is, after the second sound, as a vibratory sensation, gathers force and volume, and terminates with the decided shock of the impulse. This sign is pathognomonic and has been compared to the sensation imparted to the hand when placed upon a purring cat (*frémissement cataire*; Laennec). In some cases it can be best elicited after physical exertion, or by elevation of the arm while in the recumbent posture on the left side, or it may become more marked by causing the patient to sit up and lean forward.

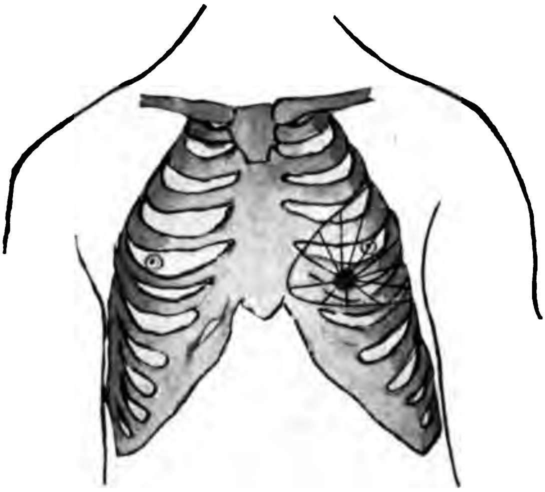

FIG. 22.—Area of the mitral presystolic murmur. The dark circle indicates the usual locality of maximum intensity, lines show the possible directions of transmission.

Palpation may also disclose an impulse at the base of the heart associated with the closure of the semi-lunar valves and observed over the second left intercostal space. This impulse is caused by

In advanced cases there may be marked anxiety and cyanosis. A common form of mitral obstruction is that observed in chlorotic young women with menstrual and nervous disorders, pallor of the countenance and blanching of the mucous membranes.

The pulse is generally regular until the heart begins to weaken unless obstruction is complicated by regurgitation or some other valvular affection. In some instances, in the earlier stages it may be somewhat empty with loss of pressure. Later, as the disease advances, there will be decided changes and the vessels will be more empty and compressible. This will be followed, as the failure in compensation increases, by irregularity in rhythm and inequality of the pulse-waves. Some of the heart pulsations may fail to reach the wrist. There may be two or three pulsations, then a pause, then, again, pulsations. In some cases there is one beat at the pulse for two at the heart, the contraction of the left ventricle at every alternate systole being unable to raise the aortic valves, and on auscultating it will be found that there is no aortic second sound with the alternate systole. Again, in some cases the pulsations at the wrist are apparently independent of the rhythm of the heart.

Physical Signs.—*Inspection* may disclose little or no displacement of the apex-beat. But when hypertrophy of the right ventricle is excessive, or associated hypertrophy of the left is present, it is displaced to the left and downward, or it may give evidences of its presence to the right of the sternum, but generally it is not found far from its normal position. There may be a strong epigastric pulsation from the right ventricular hypertrophy, and in thin persons an impulse may be visible in the third and fourth interspace to the left of the sternum. The apex impulse, as the disease advances, is liable to weaken and sometimes disappears. Thus when compensation is complete nothing abnormal may be observed, but when it is broken the apex-beat may be feeble or invisible. In children there may be some prominence of the inferior extremity of the sternum and of the fifth and sixth costal cartilages from enlargement of the right ventricle.

Palpation shows diffusibility of the apex-beat. The heart imp
may be felt to be more forcible over the lower portion of the stern
and along its right border, also, occasionally, to the left of the s

occur later with a pause before and after, that is, meso-diastolic. When the murmur is presystolic it is said to arise from the contractile force of the auricle propelling the blood through the contracted orifice, when late diastolic, from the vigor of the right ventricle, when meso-diastolic, from the aspirative force of the left ventricle.

As a rule, anomalous or broken murmurs may be observed in conditions which at times manifest murmurs of a more typical character.

Another characteristic is accentuation of the pulmonic second sound due to backward pressure in the pulmonary artery and ventricular hypertrophy.

Reduplication of the second sound is also heard at the base in which the pulmonic predominates. When present it is audible with the greatest intensity in the second or third left intercostal space, although sometimes it may be heard over the apex, a condition difficult to explain. According to Sansom it belongs to the earlier periods of mitral obstructive lesions, while Potain, on the other hand, has observed it in the latter stages. Two explanations are given for the origin of this phenomenon, namely, that it is a sound of tension produced by the rush of blood into the ventricle being more sudden and forcible than normal from increased pressure in the left auricle due to the obstruction at the mitral orifice, the blood passing to the ventricular side of the curtains of the mitral valve causing them to bulge and thus give rise to a sound of valvular tension, which follows the first sound of the heart and causes an apparent doubling, or that it arises from absence of perfect unison in the action of the aortic and pulmonic valves, in which the pulmonic sound predominates. The reduplicated sound is liable to present variations in intensity and disappear and reappear.

Mitral obstruction is further characterized by weakening or inaudibility of the aortic second sound at the apex.

Another feature is the variable appearance of the murmur; it may disappear upon the onset of some pulmonary or other complication, when upon the subsidence of these conditions it will reappear. Again, when the heart has become weak it may be no longer audible, but when the organ has regained tone it may again be heard.

increase of the blood-pressure within the pulmonary circuit, and is of both diagnostic and of therapeutic importance.

Percussion in some cases may show no change, in others, and more commonly, it may show increase of the area of dulness mainly to the right of the apex, from dilatation of the right ventricle, and upward along the left sternal border from dilatation of the left auricle. Changes in the dimension of the heart do not bear the same direct relation to the extent of valvular defect in mitral obstruction as in other affections of the valves; the signs elicited by percussion are therefore of less importance.

Auscultation reveals in cases of obstruction of the mitral orifice, unattended with marked insufficiency, disturbances of the right side of the heart or other complications, a harsh vibratory presystolic murmur which terminates with the first sound and coincides with the presystolic thrill. Its point of maximum intensity is at the apex and to its inner side, between it and the left border of the sternum. The area of transmission is usually limited to this locality, though sometimes it may be transmitted somewhat to the left. A feature is the manner in which the murmur runs up to and terminates with the first sound, which becomes sharp and abrupt. For this reason it is not always easily separated from the latter, but it may be distinguished by its qualities if not by its time. In some instances it may exhibit variation, being short and low in its tone.

While the murmur belongs to the diastole it may vary somewhat in its relation to this period. It is generally characterized as presystolic, that is, occurring during diastole just before the systole, and corresponding to the systole of the left auricle. It usually occupies the latter half of the diastole, but it may be shorter or longer, or may occupy the entire diastole, or vary in its relation to the diastole. That is, it may begin almost immediately after the second sound and be prolonged throughout the entire diastole; it may also be increased in force toward its end and terminate in a sharp, snapping sound. In some instances these features may be observed to be simultaneous in action with the apex impulse as recognized by palpation, in others they may be found to occur immediately after the first shock of the impulse. Again, the murmur, instead of being prolonged or presystolic, may be heard as a short sound following immediately the second sound, that is, early diastolic, or it may occur later with a pause before and after, that is, meso-diastolic. When the murmur is presystolic it is said to arise from the contractile force of the auricle propelling the blood through the contracted orifice, when late diastolic, from the vigor of the right ventricle, when meso-diastolic, from the aspirative force of the left ventricle.

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Owing to these variations and differences, the physical signs of mitral obstructive lesions as disclosed by auscultation may be considered more advantageously by dividing the subject into stages. Broadbent recognizes three stages, but it is often difficult to clearly define the dividing line between the second and third stage.

In the first stage the signs are those of the presystolic murmur, heard at the apex in conjunction with the presence of the first and second sound, the persistence of the latter marking this period of the affection. As a rule, serious complications do not appear.

The second stage is distinguished by the disappearance of the second sound at the apex and by the changes in the first sound, which becomes sharp and abrupt, appearing somewhat like the second. The disappearance of the second sound is due to the decrease in the amount of blood-supply in the aorta, arising from diminished supply to the ventricle; in consequence of which there is a feeble recoil against the closed aortic valves, and to the overtopping of the left ventricle, which is the principal conductor of the sound, by the enlarged right auricle and ventricle.

The change in tone of the first sound is attributed by Broadbent to sudden tension of the muscular walls and the shortened systole of the left ventricle, which this writer explains, is as follows: "During diastole, on account of the contraction of the mitral orifice, the left ventricle does not receive sufficient blood to fill it completely, so that at the beginning of systole its cavity is not fully distended; therefore, as soon as its walls begin to contract they meet with no resistance, and in consequence close rapidly; in doing so they abruptly encounter the blood in the cavity, and as a result the muscle is suddenly tense."

The third stage is marked by the inaudibility of the presystolic murmur. The second sound having already disappeared, the only sound heard at the apex, therefore, is the first, which is abrupt and sharp, as mentioned. The cause assigned by some writers for the loss of the murmur is the presence of tricuspid insufficiency, which reduces the blood-pressure to such an extent that the force which propels the blood through the mitral orifice is not sufficiently strong to produce a murmur. The failure of the tricuspid valve being followed by regurgitation into the right auricle renders it impossible for the right ventricle to maintain the former amount of pressure in the pulmonary vessels and left auricle, hence the reduction in propelling power. The phenomenon of loss of presystolic murmur is usually regarded as of serious significance, but this is not necessarily so.

Diagnosis.—The recognition of mitral obstruction when the physical signs are present is not attended with difficulty. The presystolic thrill, increase of præcordial dulness upward and to the right, the presence of a murmur, presystolic or diastolic near the apex-beat, localized and audible rather with the second sound than with the first, or, more accurately, just before the first, and terminating with it—the murmur itself being harsh and vibratory—and decided accentuation of the pulmonic valves, are characteristics which are diagnostic.

When these features are absent, as they not infrequently are, a positive diagnosis cannot always be made. There are, however, certain signs which may be considered as presumptive evidence of the lesion. These are, harshness in quality of the first sound, especially if accompanied by doubling of the second sound, and strong accentuation of the second pulmonic sound. Some writers regard these signs, in the absence of all others, as sufficient to establish the diagnosis of mitral obstruction, but this is scarcely warrantable.

When uncomplicated a mitral obstruction murmur is not liable to be confused with other adventitious sounds. Its harshness, presystolic rhythm and limited area of transmission distinguish it from insufficiency. But the fact that the lesion seldom occurs alone and generally in association with insufficiency adds a different aspect to the condition, and often the distinct recognition of the two murmurs is attended with difficulty.

The presence of a soft blowing systolic murmur synchronous with the carotid pulse with its area of transmission to the left as far as the axilla, usually readily recognizable on account of its prominence, obviously points to regurgitation. If an obstructive lesion is also present, auscultation to the right and above the apex discloses a presystolic murmur audible over a limited area. Furthermore, areas may frequently be located where the bruit is continuous, occupying a portion of the systole and diastole, especially when compensation is failing. When a presystolic murmur is heard at the apex, in connection with double aortic murserious, for the reason that compensation is less readily established and not so efficiently sustained.

The most important evidences upon which judgment as to the future can be based are those obtained by a careful examination of the heart-sounds. As long as the second sound is present at and beyond the apex, the probabilities of the appearance of symptoms are remote. When, however, the second sound is lost, that is, when the second stage is reached and the lesion is more advanced, there may still be absence of symptoms, but the heart may show its weakness by a marked susceptibility to extraneous influences, such as fatigue on slight exertion, gastric disturbances and mental symptoms. In short, the favorable state of the patient may be more apparent than real. With care, however, these cases may continue comfortable for a long period.

In the third stage the danger of breaking up of compensation and failure of the heart is much greater. Venous engorgement, pulmonary congestion and over-distention of the right side of the heart may be easily excited. On the other hand, patients with the auscultatory signs peculiar to this stage may, with care, live a considerable time free from serious symptoms.

A feature of mitral obstruction is that conditions of serious embarrassment of the pulmonary circulation with all their concomitant symptoms may improve and the patient recover. But attacks of this nature when repeated or if easily excited are always of very grave significance.

Treatment.—In the first stages of mitral obstruction there is rarely any demand for treatment other than the general hygienic rules necessary for those suffering from valvular defects, but greater care for the preservation of the heart muscle, the use of cardiac remedies with caution and with special regard to their possible harmfulness will be called for at an earlier period. When compensation begins to show signs of derangement and the question of therapeutic interference presents itself, there are several points which require special consideration. Bearing in mind that the first manifestations of disturbance are connected with the pulmonary circulation, attention should be primarily directed toward relief of the lungs. All influences which tend to increase arterial tension sh also be avoided, as they must result in higher pressure within heart chambers and thereby cause further irritation. The diet should be simple and free from over-indulgence. Stimulants and the too liberal use of fluids should be prohibited. The bowels should not be allowed to become torpid. Exercise may be taken as recommended for patients suffering from other valvular deficiencies and in the earlier periods a systematic development of the heart muscle may be attempted, by carefully graded exercises. Undue exertion must be forbidden.

The tendency to bronchitis and pulmonary inflammations should always lead to caution, as their presence increases the resistive force of the pulmonary circulation and places extra pressure on the right ventricle. All sudden changes of temperature and exposure should, therefore, be carefully guarded against, and when removal is possible the patient should seek a mild and equable climate.

In acute inflammation of the bronchi and lungs, cardiac remedies may often be administered with advantage even when compensation is apparently good. Here digitalis, strophanthus and convallaria will prove of value. In persons in whom mitral obstruction has been recognized special caution is necessary in regard to rheumatism. Any of its manifestations, however slight, should receive attention. In these cases mild febrile disturbances generally points to subacute endocarditis, especially in children and young adults, and rest and anti-rheumatic treatment should at once be enjoined. When hæmoptysis occurs, as a rule, absolute rest in bed is all that is necessary. The use of hæmostatics and opium is generally to be condemned.

When the liver becomes inactive, as it frequently does, even in mild cases of weakened compensation, and the tongue is coated, bowels constipated, or, if not, stools clay-colored, and possibly there is a trace of jaundice, mercurius dulcis in the first decimal trituration should be given in hourly doses of one or two grains until the bowels move, or two or three drachms each of sulphate of soda and phosphate of soda in water, or sulphate of magnesia, may be given in hot water in the morning before breakfast. When administering remedies of this nature, any excessive action should be carefully avoided as tending to further weaken the patient.

In marked obstruction digitalis is generally regarded as to be employed with caution. In the early stages it is not called for, and in the latter it is indicated only when the right side of the heart shows signs of weakness. Its administration should in most instances be preceded by the relief of the congested liver by means of purgatives. During its use the patient should always be kept under observation. It should not, as a rule, be given for a long time in these conditions. Indications for its withdrawal are slowness of the pulse and præcordial oppression. During febrile attacks it is also contraindicated.

When infarctions occur in the pulmonary artery, Sir Benjamin Richardson recommended liquor ammoniæ fortior for dissolving the coagula and liquifying the blood. The remedy also acts as a stimulant and, by rendering the bronchial secretion more fluid, as an expectorant. The dose employed was one to five minims well diluted in water, taken every half hour at first, then every hour or two until improvement, after which it may be continued every four hours for several days.

In advanced forms of mitral obstruction the pulse becomes weak, irregular and at times imperceptible, the heart pulsations violent, especially those of the right ventricle, the apex-beat almost lost, jugular pulsations marked, liver enlarged, dropsical symptoms increased, the right ventricle no longer able to contract, and a fatal termination imminent. Dyspnœa may be excessive, but not necessarily intensely so, the countenance may be pale and cyanotic and sometimes there is a reddish spot on each cheek. Under these circumstances the contracted mitral orifice may remain a fixed obstacle maintaining a steady backward pressure in the pulmonary circulation and thus make it difficult for the left ventricle to overcome the over-distention to which it is subjected. When this condition is recognized the local abstraction of blood may be resorted to by means of leeches in order to lessen the amount of blood in the veins and thereby afford the semi-paralyzed right ventricle an opportunity to recover from its over-distention and thus contract more effectively. Six or eight leeches may be applied over the liver and epigastrium. In conjunction with this measure efforts should be made to relieve the portal circulation.

The principal indication for resorting to the abstraction of blood is the contrast of the feeble pulse and the bounding heart in association with dyspnœa and threatened collapse.

After the right ventricle has been thus relieved of its immediate stress by these mechanical measures, digitalis, strychnine and other remedies as specially indicated of like nature may be administered with a better prospect of proving effective.

For further remedial measures the reader is referred to the article on general treatment.

Aortic Insufficiency.

Aortic insufficiency signifies imperfect closure of the aortic valve whereby the blood flows back from the aorta into the left ventricle during diastole. As an uncomplicated murmur it is uncommon, being generally found associated with obstruction of the aortic orifice.

Ætiology.—Arterio-sclerosis and endocarditis are the chief factors in its causation, the larger proportion of cases arising from the former. The underlying influences which produce the pathological changes are high tension, chemical irritants in the blood, acute infectious diseases and syphilis.

Undue tension may arise from any influence which occasions excessive functional activity of the heart. Prolonged and continuous muscular efforts and laborious occupations constantly augment the blood-pressure and thereby tend to gradually induce arterio-sclerosis. For this reason men in middle life who have engaged in manual labor are often the subjects of valvular disease, notably of the aortic area. For the same reason aortic insufficiency is not infrequently observed among those who indulge to excess in athletic sports, such as running, rowing, weight-lifting, hammer-throwing, etc. Hence the term "athlete's heart." The continuous immoderate use of alcoholic stimulants also tends to the same end. Frequently, alcoholic stimulants and excessive tension from manual labor are both operative in the same patient. Sometimes an aortic regurgitant murmur may arise from high arterial pressure in cases of severe exertion or in senile arterial plethora, and disappear after rest and treatment.

The presence of uric acid in the circulation acts as an irritant and likewise induces atheroma and interstitial endocarditis. Gout, therefore, especially in its chronic and irregular forms, is a potent cause. Lead poisoning, by favoring the formation of uric acid, may also, indirectly, give rise to these conditions. Alcohol acts as an irritant as well as an excitant of the heart, hence it may contribute in two ways to the causation of atheroma. Among acute infections, endocarditis arising from any of the causes previously described, especially rheumatism, proves the most frequent source of disability of the aortic valve. Lesions of this type generally belong to early life. They play, however, a much less important part in the ætiology of aortic disease than they do in the case of that of mitral, and, when occurring, will, as a rule, be observed in association with the latter.

Aortic insufficiency may, in rare instances, arise during the course of ulcerative endocarditis, but such cases usually terminate speedily in death.

Syphilis, while not a prominent element, is certainly in many instances at least a contributing cause. The association of aortic insufficiency and locomotor ataxia has led to the supposition that there is some close affinity between the two affections, but the presence of syphilis points to a common origin as the source of connection. This view, however, is not by any means universally accepted.

Traumatism is an element of importance, and is more likely to result in aortic insufficiency than in any other lesion of the valves. The valve may be ruptured in consequence of injury or strain. The left posterior cusp is the part usually affected and the injury is generally confined to that locality. Such instances, however, are not of common occurrence.

Taking into consideration the relative frequency of the lesion in regard to age and sex, it will be observed that aortic insufficiency is more common in after middle-life and in males than in the young or in females. The reason is evident from the fact that its causal influences are more operative in middle life and among men.

Morbid Anatomy.—The pathological processes are usually confined to the cusps. In some instances dilatation of the aortic orifice is the cause of insufficiency. This view, however, is challenged by some writers.

When the degenerative changes are atheromatous there is shrinkage, thickening and deformities of the cusps with patches of grayish-yellow. Sometimes a segment of the valve becomes wholly or in part adherent to the walls of the aorta. In some instances the processes break down and form ulcerations or lead to perforations. Associated with these changes the wall of the aorta beyond the orifice is often the seat of sclerosis and calcification. There may also be some dilatation of the aorta at this point which may be either general or partial. In consequence of these lesions the openings of the coronary arteries are liable to become obstructed and their walls involved in the atheromatous degeneration. This is a most important feature in aortic disease.

When the lesion arises from endocarditis there are characteristic thickening, fibrinous deposits and vegetations of various sizes situated on the surface of the cusps opposed to the blood-flow. Atheroma is liable to follow with ulceration, or, more commonly, deformities of the cusps from shrinkage or adhesions of their thickened margins. In long-standing cases there may be calcareous deposits and consequent rigidity. In some instances endocarditis may result in fusion of the cusps at the margin with the consequent production of a deformity of the orifice, often accompanied by an intense degree of hardness.

From the nature of these pathological changes it will at once be seen how the conditions which give rise to insufficiency of the aortic valve may also, in many instances, be cause of obstruction.

According to Beneke, the normal diameter of the aortic orifice at birth is twenty millimetres. This increases with the growth of the body until at the age of twenty-one it is sixty millimetres. From this time on there is a gradual increase up to the age of seventy-five or eighty when it measures from sixty-eight to seventy millimetres.

MECHANICAL EFFECTS OF THE LESION.—The reflux of blood as it pours through the aortic orifice, into the left ventricle during diastole, meets the normal current as it comes from the left auricle. The result is distention of the left ventricle which, in order to fulfill the increased demands made upon it, dilates and hypertrophies. As the ventricular muscle is powerful the degree of hypertrophy may become excessive, forming, in some instances, the *cor bovinum*. The regurgitation of blood through the aortic orifice tends to lessen the supply to the arterial system, but in the earlier periods the increased force of the ventricle compensates this deficiency. When the lesion is well established the aortic reflux causes comparative emptiness of the arteries, and the hypertrophied ventricle suddenly and forcibly expels its contents into the aorta; the result is an excessive degree of pressure during systole and an abrupt and exaggerated lowering of the same during diastole. It will thus be observed that the vessels near the heart are subjected to increased pressure, at the same time those of the periphery fail to receive the full supply of blood. In consequence of this fluctuation of pressure the arterial walls are liable to become the seat of arterio-sclerosis and atheroma, and subsequently the source of aneurysm and apoplexy.

When the left ventricle weakens additional work is at once thrown upon the left auricle which undergoes a certain amount of hypertrophy, though not well adapted to this condition. As the blood accumulates in the left auricle it impedes the free flow from the pulmonary veins. There now arises a damming back of the pulmonary circulation which leads to increased pressure in the pulmonary artery. Next follows dilatation and hypertrophy of the right ventricle which, for a time, enables the ventricular cavity to empty itself and to propel the blood through the congested capillaries into the pulmonary veins and left auricle. The right ventricle, therefore, assumes compensation to a marked degree, and it will be seen that the right ventricular hypertrophy contributes very largely to the enlargement of the heart.

As the hypertrophy of the left ventricle continues to increase it becomes, in time, sufficiently strong to empty itself and thus relieves the left auricle; in consequence the stasis of the pulmonary veins, lungs and pressure in the pulmonary artery are also relieved. As a result, the hypertrophy of the right ventricle may subside to a great extent and not reappear until the left ventricle begins to give way. In consequence of the long and continued strain to which the ventricle is subjected, in time the excessive hypertrophy leads to degeneration and loss of power. Dilatation then predominates, the mitral valve becomes incompetent either from relative insufficiency or from sclerosis, the right side of the heart becomes involved, the tricuspid valve fails, and the long train of symptoms attendant upon mitral insufficiency in its last stages ensue. As long, however, as the mitral valve remains intact, the pulmonary circulation will not be disturbed. Again, as hypertrophy increases the heart, in many instances outgrows the capabilities of the coronary arteries to furnish it sufficient nutrition. In consequence fatty and fibroid degeneration, with secondary dilatation, may develop, the onset of which may be regarded as the signal of failing heart power. Deficiency in blood-supply for the heart muscle also arises from ob-

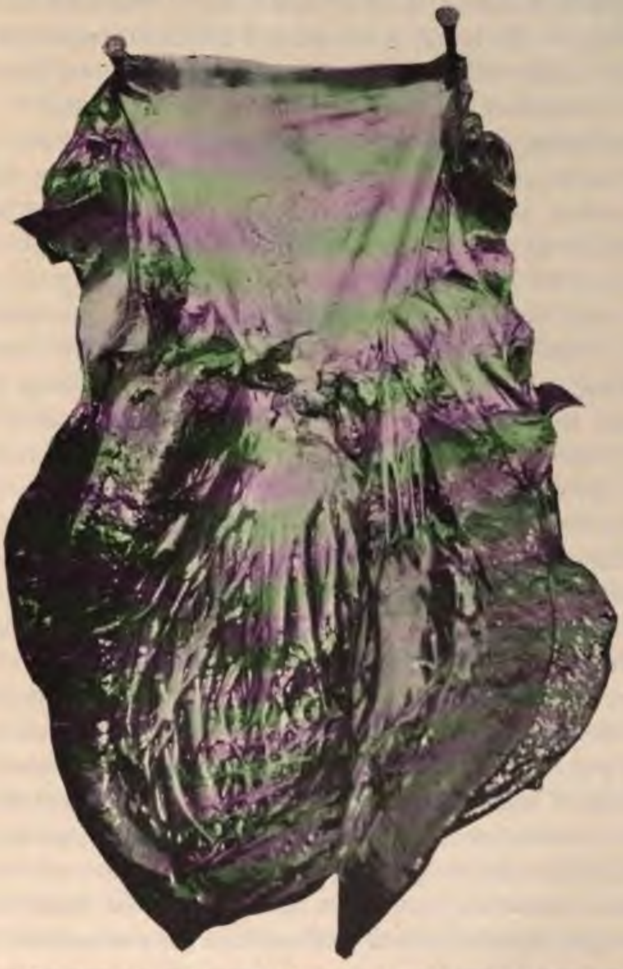

FIG. 23.—Chronic endocarditis and atheroma, showing marked degeneration and destruction of the aortic valve, calcareous deposits in the aortic area and valves, and an excessive degree of hypertrophy of the left ventricle, the walls of which are laid open.

struction of the mouths of the coronary vessels, constriction of their lumen and loss of elasticity of their walls, the result of sclerotic and atheromatous changes. But, so long as the coronary arteries are not impaired and are able to efficiently supply the heart muscle, and the quality of the blood is maintained sufficiently near the normal standard to afford the required nutrition to the myocardium, hypertrophy compensates and evenly balances the circulation.

Symptoms.—In the greater proportion of cases the onset of aortic insufficiency is insidious and the development of its symptoms gradual, as might be inferred from the ætiology. Frequently it is discovered by accident. In many instances the lesion may remain latent for a considerable period without exciting any disturbances or giving any evidence of cardiac disease, until some intercurrent illness or unusual demand is made upon the heart, when palpitation, pain and other phenomena pointing to the presence of a cardiac lesion become manifest. Under these conditions it is to be presumed that the pathological processes have covertly involved the valve structures, but being of such a mild type no visible effect is produced until additional stress is laid upon the heart.

On the other hand, symptoms of gravity may arise suddenly, generally as the result of strain or injury, which causes rupture of a valve and is immediately attended with pain, dyspnœa and serious circulatory disturbances. Cases of this nature are rare.

In the larger proportion of patients where compensation is adequate, there is absence of symptoms; in some instances, however, there is palpitation and dyspnœa on exertion, which entirely disappear when at rest. In one class of cases decided symptoms of circulatory disturbance, such as small, empty pulse, dyspnœa, cyanosis and pulmonary congestion, may attend the onset of the lesion, but after a time these symptoms subside with the establishment of hypertrophy, and the patient may continue for a considerable period in apparent good health.

As the brain is exceedingly sensitive to circulatory disturbances, patients with aortic insufficiency are liable to various cerebral symptoms. They may suffer from a throbbing sensation in the head, pulsating headache, dizziness, especially on rising from a recumbent posture, insomnia, tinnitus aurium and flashes of light before the eyes. In advanced cases there may sometimes be mental perturbation. The association of aortic insufficiency with arterio-sclerosis must be taken into consideration and, in the instance of cerebral symptoms, its possible presence in conjunction with arterial anæmia.

Dyspnœa may appear early, but it is not, as a rule, as prominent as in mitral disease. Usually it is not excessive, but may become so, especially from violent exertion or strong emotional influences. It is due, in many cases, to defective aëration from simple pulmonary engorgement arising from interference with the return flow of blood, but it may also arise from changes in the kidneys associated with arterio-sclerosis.

A sense of oppression in the præcordium and attacks of palpitation are common and frequently are the source of distress.

The countenance is pale, either as the result of peripheral anæmia or from sclerotic condition of the vessels. Dilatation of the peripheral vessels may occasionally be attended with hot flushes and profuse perspiration.

Pain is more prominent in aortic insufficiency than in any other form of valvular defect, on account of its intimate connection with atheroma and consequent implication of the coronary arteries. This important, and at the same time serious, symptom may vary from a dull, aching sensation to the intense agony of angina pectoris: It may be confined to the præcordial region or may extend down the arms, particularly the left. It may also involve the shoulders, nape of the neck and head.

Abnormally strong, violent pulsation of the arteries near the surface is a characteristic feature. In many instances it is excessive. The carotids are obviously the most prominent, but the facial, temporal, subclavian and the arteries of both the upper and the lower extremities may also show exaggerated movement. Ophthalmic examination may disclose pulsation of the vessels near the fundus. The patient often complains of the throbbing of the carotids and a sensation of fullness in the head.

The peripheral circulation frequently presents important and peculiar features in the form of venous and capillary pulsation.

When the pulsation is violent a general venous pulse may be observed. To observe this sign, direct the patient to extend the hand and let it drop at the wrist, the viens on the back of the hand will then fill out and pulsate with a gentle movement.

Capillary pulsation of the cutaneous vessels was first pointed out by Quincke and Ruault. It may be observed by applying friction to any portion of the normal skin, the hyperæmia thus produced becomes pale with each diastole and reddens with each systole. The same sign may sometimes be seen in the lacunæ of the nails, and around urticarious patches. The cause of this phenomenon is not difficult to find. The great volume of blood thrown out by the excessively dilated and hypertrophied ventricle suddenly dilates the vessels, but, on account of the falling back of the column of blood through the aortic orifice, this pressure is not maintained, so that after each pulse-wave the vessels as suddenly collapse.

Capillary pulsation, however, is not alone peculiar to aortic insufficiency, inasmuch as it may occur in other conditions when the arterial tension is low, as in tuberculosis, typhoid fever, neurasthenia and pernicious anæmia.

The pulse presents special features. In the interval between the beats it becomes completely empty, then a wave comes with an abrupt and sudden gush filling the vessel completely. Each pulsation is of short duration and falls away from the fingers as soon as it reaches them. The sensation imparted is generally compared with that of a water hammer, hence the term "water-hammer" or collapsing pulse. Sometimes it is called after Corrigan, who first described it.

In order to observe this phenomenon to the best advantage the patient should be placed in the recumbent position with the hand at about the level of the shoulder, that is, above the level of the heart. When the hand is hanging down the pulse remains full, as the pressure is more evenly sustained on account of the volume of blood above it. The feature of this pulse is the contrasting conditions of emptiness and supernormal fullness.

The exaggeration of the undulations and collapse of the pulse-wave is a striking feature of aortic insufficiency. But it is not observed when aortic obstruction is present, which interferes with the collapse of the pulse-wave, in the latter stages when the force of the systole is weakened, or when aortic insufficiency is the result of excessive degeneration, in which case there is loss of elasticity in the vessels and the amount of incompetence of the valves is not great.

The exaggeration of the pulse-wave in aortic insufficiency, when present, may serve as a guide to the extent of the lesion, the more marked the undulations the more extensive the regurgitation.

Another feature of the pulse in aortic insufficiency is the delay between the heart-beat and the radial pulsation. This is due to the absence of a continuous stream in the arteries, to the collapsed state of the vessels between pulsations, and to their loss of tone.

A double pulsation, pulsus biferiens, is sometimes observed, especially when insufficiency is associated with obstruction. This is a peculiar double beat, but more readily detected by making moderate pressure on the artery, somewhat less than is necessary to elicit the collapsing character. The cause is due to a double systolic impulse which occasionally may be felt or heard over the heart and is frequently audible in the carotids. Extra exertion will make it more readily observed when otherwise it would not be discernible.

In the later stages of failing compensation the pulse becomes irregular. At first there is less force in the pulsations which occur irregularly, while the intervals are longer. As the weakness advances these features become intensified, but the characteristics of aortic insufficiency may be distinguishable.

The symptoms which arise when compensation fails present two general types. In the first instance the conditions are due to the inability of the left ventricle to sufficiently sustain the capillary circulation, in consequence the symptoms are characterized by persistent sleeplessness, intense restlessness and difficult respiration. The countenance is pale and anxious, with an expression of much suffering. Dropsy is not marked, but there may be some effusion into the pleural cavity and some œdema of the base of the lungs. Sudden death is liable to occur even in spite of apparent temporary relief.

In the second type the condition results from failure of the right ventricle secondary to that of the left. Here there is dropsy, hepatic enlargement, jugular pulsations, general venous stasis and the phenomena which attend the later stages of mitral insufficiency. This condition may also terminate suddenly in fatal syncope, but such a contingency is not so liable to arise as in the former instance.

Physical Signs.—*Inspection* generally shows no change in the form of the chest except in young patients and those with small chests in association with excessive cardiac enlargement in whom there may be some bulging of the wall. In most cases the apex-beat is lower and more to the left than normal. It may be down as far as the sixth or seventh intercostal space and beyond the mammary line. The force of the impulse is stronger than normal and generally diffuse. As enfeeblement of the left ventricle increases it becomes more undulating.

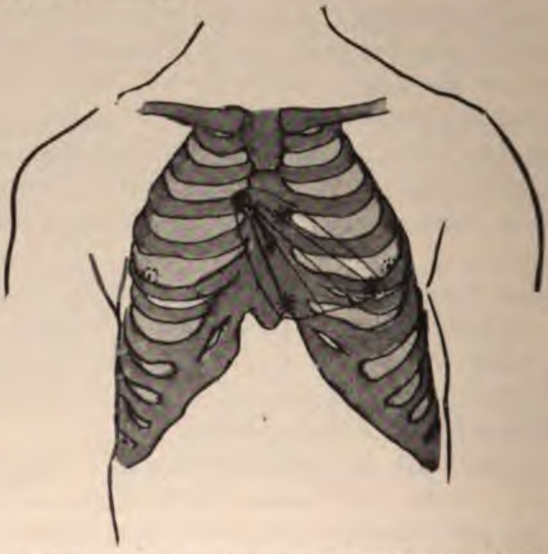

Fig. 24.—Area of the aortic diastolic murmur. The black circle indicates the usual locality of the maximum degree of intensity, the stars occasional, but not uncommon, areas of intensity, the lines the directions of transmission.

Palpation discloses increased force of the impulse. A heavy impulse is usually felt showing an increase of the diastolic recoil as well as of the systole. A sensation suggestive of a thrill may, in some cases, be observed at the apex. This is due to the increased energy of the heart. Sometimes a diastolic thrill may also be felt at the apex and above the sternum and over the whole præcordium. This is owing to the vibration caused by the reflux of blood from the aorta.

Percussion gives increase in the area of dulness. In some instances it may extend downward as far as the seventh rib, and laterally as far as the left axillary line. The extent of the area depends largely upon the degree of hypertrophy of the left ventricle. In the later stages there may sometimes be increase of dulness, upward and to the left of the sternum from enlargement of the left auricle. When the right side of the heart has become enlarged there may be increase of dulness to the right.

Auscultation discloses the presence of a diastolic murmur with characteristics which may vary widely. In most cases the murmur is heard to the best advantage in the right second intercostal space close to the sternum, or at the junction of the second costal cartilage with the sternum. The area of its transmission is downward along the side of the sternum or obliquely toward the apex. It is also described as heard with the greatest intensity over the right half of the sternum above the level of the fourth costal cartilage and sometimes entirely to the left of the sternum in the vicinity of the third intercostal space. Again, it may be audible only at the apex. These variations are difficult to explain satisfactorily. Sir William Foster says in regard to the presence of a diastolic murmur of aortic origin, audible at the apex, that it depends "on the regurgitation taking place through incompetency of the posterior segments at its right angle or through perforation of the cusp, so that the regurgitant blood column falls upon the anterior curtain of the mitral valve. The vibration produced, notwithstanding the lesion is of aortic origin, occurs in the mitral area and is heard at the apex.

The phenomenon of a presystolic murmur in aortic insufficiency simulating mitral stenosis was first discovered by the late Dr. Austin Flint who, about thirty-four years ago, published the report of two cases. In both of these patients during life a presystolic murmur was present, but after death, on examination the only valvular lesion found was that of aortic insufficiency, the mitral valve being free from stenosis. This condition has been the subject of wide discussion as well as of divergent opinions as to its cause. Sansom offers the following explanation: "It may arise in two ways: either the lifting force of the current impinging upon the under surface of the great mitral curtain might so obstruct the current from the auricle as to create a *de facto* impediment at the end of each diastole; or the vibration might be directly communicated by the regurgitant stream from the aorta to the great mitral curtain. The nearness of the posterior segment of the aortic valve (diseased perhaps so that the morbidly produced orifice in diastole presents a ragged or fringed border) to the mitral flap may well account for such vibrations. These vibrations may attain only to the intensity and rapidity sufficient to cause murmur somewhat late in the diastolic period. The force of the auricular systole would necessarily amplify and intensify such vibration of the free edge of the flap until the commencement of the ventricular systole or tug of the chordæ tendineæ stopped them, bringing both curtains of the valve together and completely close the auriculo-ventricular orifice."*

It is possible for the lesion to be present and the murmur absent This may occur when there is extreme stenosis and in association with mitral disease.

The character of the murmur is usually soft and blowing and of great intensity. The second sound is generally lost, but may be distinctly audible when the cusps are slightly curled or distorted or when only one cusp is involved and regurgitation slight. In rare cases it is harsh and vibratory. A soft murmur is generally confined to the sternum, but a harsh murmur may be widely diffused over the chest. The murmur may be short or prolonged, in the latter case, especially when heard during the latter part of the diastolic pause, there may be some difficulty in distinguishing it from that of mitral stenosis.

Attention is called to the statement of Osler that in a large proportion of cases of aortic regurgitation a murmur is propagated upward in the vessels of the neck, and which is not due to an actual stenosis, but to a roughening of the segments or the intima of the arch.

Diagnosis.—The recognition of aortic insufficiency is not, as a rule, difficult. The diastolic murmur at the right margin of the sternum, the excessive hypertrophy of the left ventricle, the peculiar collapsing character of the radial pulse, the presence of the pulsations of the arteries near the surface and in some instances, capillary and venous pulsation, are the essential features which indicate the presence of the lesion.

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* System of Medicine, T. C. Allbutt, New York, 1897.

Generally there is little likelihood of error in establishing the diagnosis.

Aortic insufficiency may be distinguished from mitral obstruction by the presence of marked hypertrophy of the left ventricle and by the fact that the murmur, with rare exceptions, is heard with its maximum intensity in the vicinity of the base of the heart. The presence of the Corrigan pulse and the excessive pulsations of the arteries near the surface point definitely to the exclusion of mitral obstruction.

The diastolic mitral murmur is heard with its greatest intensity near the apex-beat and is of a harsh, vibratory character. These two features generally render error impossible, especially in uncomplicated cases when taking into consideration the other features which characterize aortic insufficiency.

From lesions of the pulmonary valve the aortic murmur is distinguished by the fact that it is heard with its greatest intensity upon the right side of the sternum, while the pulmonic is heard at the left. In addition the extreme rarity of the pulmonic murmur should be taken into consideration. The pulse which characterizes aortic insufficiency is absent in other valvular diseases.

The previous history may also lend additional aid, especially of those conditions which result in arterio-sclerosis, such as alcoholism, gout and syphilis.

When pericarditis is seated principally in that portion of the pericardium lying directly over the aorta, a functional sound may be developed which simulates an aortic regurgitant murmur. The history of the case, the presence or absence of the peculiar pulse of the aortic lesion and of the characteristic hypertrophy will enable one to make a differential diagnosis.

Aneurysm of the thoracic aorta may prove a source of difficulty in diagnosis. In some instances the two lesions may co-exist, in which case the dilatation of the aorta is usually the cause of the valvular insufficiency.

In aneurysm the heart is displaced downward and to the left, thus simulating the increase in size which accompanies aortic insufficiency. The arch of the aorta is the most frequent seat. In this instance the characteristic bruit and thrill are observed above to the left and in the notch of the sternum. When the ascending aorta is the seat of aneurysm the bruit is to the right of the sternum extending over a wide area to the right. Furthermore, the area of dulness in advanced cases of aneurysm is beyond the area of the præcordium. The aneurysmal bruit is diffused over a wider area and becomes feeble as the heart is approached. It corresponds to the systole, while that of aortic insufficiency corresponds to the diastole. Aneurysm never causes the peculiar collapsing pulse nor the double sound of distention and collapse of aortic insufficiency. Again, tracheal tugging is present in aneurysm and absent in aortic insufficiency.

Prognosis.—While the patient may suffer little or no inconvenience and may live for years, as a rule aortic insufficiency is not favorable to long life. Of valvular affections it is generally recognized as the one above all others most liable to terminate in sudden death. It is, however, usually long latent and compensation with care may be maintained for many years.

In forming an opinion in regard to the prognosis, the factors which must be taken into consideration are the origin and type of the lesion, the age of the patient at which it was acquired, the presence or absence of other valvular defects, the condition of the arterial walls and kidneys and the state of the general nutrition. The habits, occupation and circumstances of the patient must also be borne in mind.

When aortic insufficiency arises from endocarditis the prospect is more favorable than when due to arterio-sclerosis, likewise the absence of other valvular lesions tends to lend a more hopeful view.

A condition which may be regarded as affording the fairest prospect is that when in association with the diastolic murmur the aortic second sound is clearly audible in the neck, the collapsing character of the pulse is moderate, and the dilatation and hypertrophy not excessive.

On the other hand, when the lesion is sufficiently advanced to obliterate the second aortic sound at the neck, and the collapsing character of the pulse is marked, the dilatation and hypertrophy very decided and the cardiac contractions feeble, the outlook must be regarded with apprehension. At the same time, the patient may, with care, live for years and may even be capable of undertaking exercise and work ; but sooner or later the symptoms which characterize failing compensation will appear.

Age in all instances is important. When the affection is acquired in young subjects, the chances of the heart being able to establish compensation are favorable sometimes, even in the face of adverse circumstances. But in youth, as well as in later life, when decided symptoms appear, there is always danger. On the other hand, when the onset of aortic insufficiency occurs in later life the power of the heart to undergo compensatory changes is limited and the prognosis must be considered serious irrespective of the source of the lesion, for after middle life the nutrition of the heart is not well sustained and there is always a tendency to augmentation of resistance in the peripheral circulation.

When aortic insufficiency is associated with degeneration of the arterial walls and the course of the lesion points to degenerative changes, the prognosis is not hopeful, especially as the coronary arteries are liable to become involved. Should angenoid attacks appear, additional gravity is lent to the situation.

Disturbances of the renal circulation, by throwing additional stress upon the heart, are obviously unfavorable, while the state of the general nutrition influences the heart favorably or the reverse, according as it is well or feebly sustained.

It has been claimed by eminent authority that recent cases of aortic insufficiency sometimes result in recovery. Leyden states that in a case of traumatic origin the lesion was repaired by a fibrous cicatrix. Potain claims also recovery in recent cases. Such instances, however, must be regarded as rare.

Treatment.—Notwithstanding the unfavorable phases which characterize the prognosis of aortic insufficiency, much can be done to prolong life.

A patient by some imprudent act may suddenly terminate his life, who with proper precaution might have lived, as far as the heart was concerned, for many years.

In recent cases when the lesion is recognized soon after its onset, complete and absolute rest in bed should be advised until compensatory hypertrophy has become established. During this period there is danger of fatal syncope. The patient should remain in bed from five to eight weeks, then rest in the recumbent posture for the same length of time. In the case of children they should be kept from school and active exercise for a year, while adults should refrain from business for a corresponding time.

At this stage remedies must be used with caution, for it should be remembered that it represents a period of compensatory adjustment. If the action of the heart is very tumultuous, aconite in dilution, lycopus, cactus grandiflorus, ignatia, kalmia and spigelia may be administered as symptoms indicate.

When compensation has become established there is no special demand for treatment, but caution in regard to habits of living and exercise should be enjoined. The risk of sudden death in patients suffering from aortic disease should always be remembered in laying down rules. Over-exertion, running for a train, running up stairs, excessive mental strain or excitement should be strenuously avoided.

When the pulse loses a beat occasionally and its collapsing character becomes more marked and the apex-beat is diffuse, again rest should be advised. In cases associated with sclerosis the iodides are of service. Arsenicum iodide may prove useful, also potassium iodide in five-grain doses three times a day. Strontium iodide or the syrup of hydriodic acid may be advantageously substituted for the potassium, as they are more acceptable to the stomach. These remedies require to be continued for a long time.

For palpitation and the throbbing sensation, bromide of soda often proves effective in ten-grain doses several times a day.

When angenoid symptoms appear the remedial measures discussed under agina pectoris should be employed.

When compensation breaks down the treatment will vary according as to whether it fails from inability of the left ventricle to sustain the capillary circulation, or from backward pressure in the veins, due to failure of the right ventricle arising secondary to that of the left. In the first instance efforts should be directed towards maintaining the strength and increasing the nutrition of the heart. Here stimulating remedies will be indicated, such as strychnia, glonoin and ammonia. Digitalis, if used, should be administered with caution. Strophanthus may prove a suitable substitute. For the intense restlessness and distressing insomnia often observed in this type of cases, ignatia, passiflora incarnata, belladonna and the bromides are of service. Remedies of the hypnotic class with a tendency to depress the heart are contraindicated, especially chloral.

When other measures fail, morphine hypodermically may sometimes be given with pleasing results.

When the symptoms are of the second type the condition is that of mitral disease in association with the aortic lesion. There may be dropsy, enlargement of the liver, pulsation of the jugulars and all the symptoms attendant upon the last stages of mitral insufficiency. The treatment, therefore, should, for the most part, be the same as for that lesion. The portal circulation should be relieved by mild aperient doses of calomel, then digitalis and diuretics can be administered to advantage.

The use of digitalis under these conditions should be limited to the period of dropsy and the more urgent symptoms of venous stagnation. Aside from this its administration is dangerous.

Aortic Obstruction.

Aortic obstruction is a narrowing or constriction of the aortic orifice which impedes the flow of blood from the left ventricle into the aorta. It generally occurs in association with insufficiency, as its development is frequently followed by more or less incompetency of the valve.

Ætiology.—The origin of the lesion is dependent upon the same causes which give rise to insufficiency. The same factors which lead to one lead to the other. It may occur at any age, but is more commonly observed after middle life, more so than aortic insufficiency. Like insufficiency, it is frequent among those who engage in severe manual labor, especially when long continued; for this reason it is found more often observed in men than in women.

Endocarditis in its various forms and manifestations is the source of a certain proportion of cases, but the most frequent cause is arterio-sclerosis and atheroma. Typical cases are found associated with extensive calcareous changes in the arteries in old men. In some instances a congenital origin may be recognized.

Compression from without, as from an enlarged gland, and thickening of tissue below the annular ring in the ventricular septum, conditions of rarity, have been recorded as causes of aortic obstruction.

Morbid Anatomy.—The seat of the obstruction may be at the border or on any portion of the arterial surface of the valves, at the lines of their attachment to the aorta, and in that portion of the aorta which is in immediate proximity with the valves.

In cases arising from arterio-sclerosis the structural changes which characterize this form of degeneration destroy, to a greater or less degree, the mobility of the segments, which become thickened, indurated, contracted and calcified. In some instances calcareous deposits are found in the sinus posterior to the valve. In consequence of these changes the valve cusps stand out as obstructions to the blood-flow and at the same time encroach upon the orifice. The aortic ring may be the seat of similar changes which result in constriction of its orifice. In this instance the leaflets may remain unaffected by the pathological processes. In many cases the lesion is simply a part of a general arterio-sclerosis which usually manifests itself to a more marked degree in the thoracic aorta, and which involves the aortic area by extension downwards. The coronary arteries may share in the sclerotic process as in the instance of insufficiency; when this is the case the heart walls may undergo degenerative changes. In rare instances myocardial degeneration of an atheromatous type induces stenosis of the aortic orifice.

When the pathological change arises from endocarditis, in a certain proportion of cases the infection may primarily be located in the aortic area, but in the majority it proceeds by extension from its more usual seat in the mitral valve, so that the condition may for the most part be considered a secondary inflammatory process. Vegetations are found on the surface of the valves, together with thickening and contractions of their margins. They vary in size from small granulations to large, exuberant, cauliflower-like deposits. In some cases the valve segments become adherent and fused to such a degree that they seriously impede the current of blood.

The contraction varies both in degree and form. In some instances the orifices may be so narrowed as to only admit of the introduction of a quill. It may also assume different shapes, being irregular, triangular or slit-like. Sometimes the fusion of the cusps forms a kind of funnel which protrudes into the aorta.

When the aorta is the seat of a sclerosis there is frequently dilatation which causes bulging on the concave side of the vessel.

The walls of the heart generally shows hypertrophy without excessive dilatation. This condition may continue for a long time, but eventually gives way to decided dilatation.

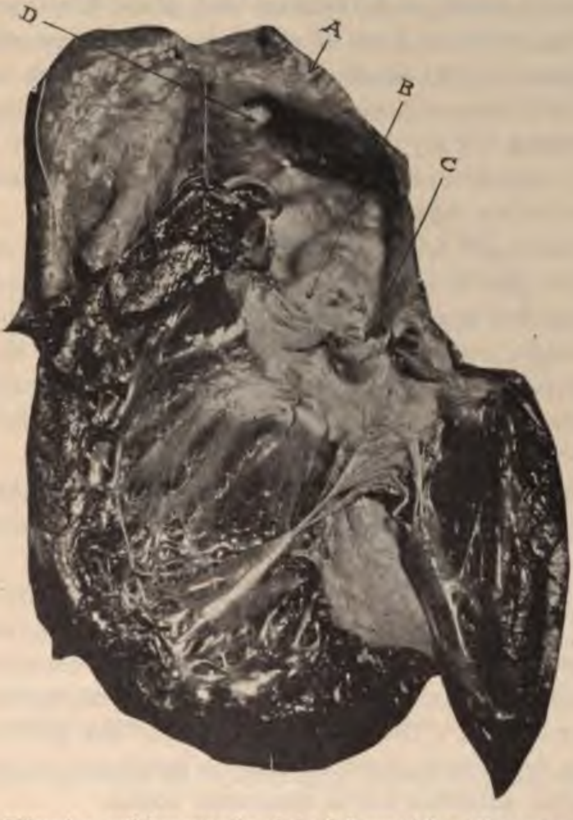

Fig. 25.—Dilatation and hypertrophy of the left ventricle. The aorta, A, is opened and stretched over the base of the heart, showing atheroma of the vessel and the aortic area, especially above the valves and around the coronary arteries, B, C, also stenosis of the aortic orifice, C, with slight aneurysm of the ascending arch which was diagnosed during life, D.

Mechanical Effects of the Lesion.—The first effect of the obstruction is to require more force from the left ventricle in order to propel the blood through the contracted opening. This causes hypertrophy of its muscular walls, a change which takes place gradually, corresponding to the advance of the lesion, but never to the extent which occurs in the instance of aortic insufficiency. Hypertrophy in time gives way to dilatation and as a result mitral insufficiency follows. Sometimes the extreme tension to which the walls of the ventricle are subjected induces sclerotic changes in the mitral area. After failure of the mitral valve there ensues dilatation of the left auricle and damming back of the flow from the pulmonary veins with the train of characteristic changes which arise in consequence of this condition. The dilatation of the right ventricle which follows will, for a time, assist in compensation.

Symptoms.—Many persons in whom compensation is complete in no way experience disturbances and may enjoy life oblivious of any trouble of the heart.

When symptoms appear, in the greater proportion of cases, they develop after middle life and in association with arterio-sclerosis.

Dyspnœa may attend slight exertion or come on without effort, but it is not as pronounced as in mitral insufficiency. In many instances there is a sensation of substernal oppression rather than a true dyspnœa. This is due to the overworked condition of the left ventricle.

The countenance shows little change, possibly a slight paleness, but as the effects of obstruction become more decided there is pallor and anæmia.

Pain is not, as a rule, as excessive as in aortic insufficiency, but it may, at times, assume the intensity of a true angina pectoris.

Variation of the blood-pressure is common, sometimes above, sometimes below normal, notwithstanding aortic stenosis in itself has little effect upon it. This is explained by the fact that the blood-pressure is dependent upon the energy of the heart and the amount of peripheral resistance which necessarily varies.

The pulse may show characteristic features. It is small and slow, striking the finger gradually, the pulsus tardus which may be said to belong to aortic obstruction. The artery is for the most part small and full between the beats. This change in the pulse is due in part to the slow distention of the arteries which results from the contraction of the aortic orifice, and in part to the rigidity of the vessels commonly associated with this lesion. The slowing of the heart action may be attributed to loss of muscular tone from faulty nutrition arising from disease of the coronary arteries.

When the apex-beat is strong it appears forcible in contrast to the small volume of the pulse. A large, sudden pulse does not belong to aortic stenosis unless there is an accompanying regurgitation. The question of the pulse, therefore, is of diagnostic value.

PHYSICAL SIGNS.—*Inspection* shows the apex-beat displaced downward and to the left, owing to the left ventricular hypertrophy. In some instances when emphysema is present it is imperceptible. Sometimes there is bulging of the præcordium and visible pulsation in the third, fourth and fifth intercostal spaces. When arterial sclerosis is present the vessels may be seen to stand out prominently.

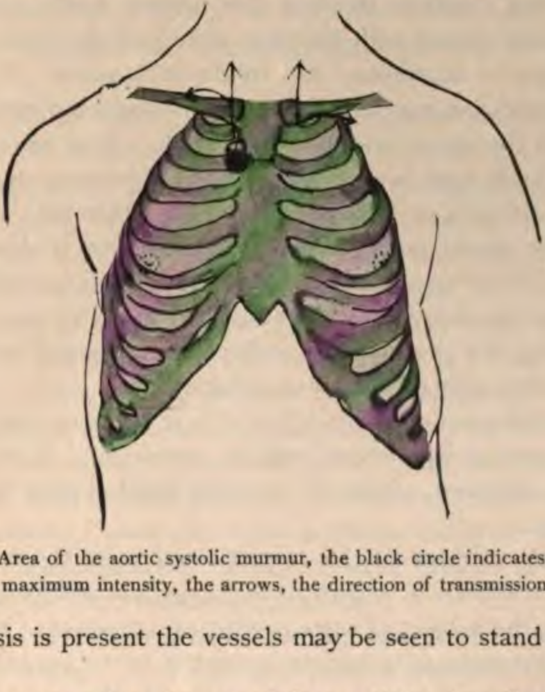

FIG. 26.—Area of the aortic systolic murmur, the black circle indicates the point of maximum intensity, the arrows, the direction of transmission.

Palpation recognizes increased force of the impulse unless the condition is associated with emphysema, in which case the movements are lost. When hypertrophy is marked a decided systolic thrill is perceptible, with its greatest intensity in the aortic region. Sometimes it is conveyed to the apex and over the whole præcordial space, but generally it is confined to the vicinity of the upper part of the sternum.

Percussion elicits dulness to the left and downward due to enlargement of the left ventricle. There may also be some increase of dulness upward if there is hypertrophy of the left auricle. The increase of the area of dulness is by no means as extensive as in the case of aortic insufficiency.

Auscultation demonstrates the characteristic features of aortic obstruction in the presence of a systolic murmur, heard in the great majority of cases with the maximum intensity in the second intercostal space at the right of the sternum. The murmur may be at times audible, with its greatest intensity at the level of the third rib to the left of the sternum. It is produced by the propulsion of blood by the ventricle through the narrow aortic opening. It begins synchronously with the first sound of the heart and may either replace or accompany it. Its duration varies. It is generally prolonged, but may be short. It is conducted upward along the arch to the great vessels of the neck. It is also frequently heard along the right border of the sternum downwards, sometimes as far as the fourth or fifth interspaces on either side of the sternum. This occurs more especially when there is dilatation and elongation of the aorta. The murmur may also be audible at the apex, being lost over the right ventricle. It is very generally conducted along the great vessels and may occasionally be audible in those of both upper and lower extremities.

It is sometimes loud, and when this is the case can be heard over the chest posteriorly as well as anteriorly. It may also be rough and vibratory, while, on the other hand, it may be mild and even musical.

The aortic element of the second sound in aortic obstruction is of importance both in regard to the diagnosis and prognosis. It is, however, the subject of some conflicting statements. On account of the hypertrophy it would be expected to be accentuated, but generally it is weakened in intensity owing to the loss of flexibility in the valves and the lack of the sudden recoil which follows the more tardy distention of the arteries.

The systolic sound in the mitral area is usually increased in intensity, prolonged and lower in tone, unless replaced by a murmur, on account of the increased force of the left ventricle.

The presence of a heaving and prolonged ventricular impulse, firmness and rigidity of the arteries in association with feebleness and a muffled second sound indicates that the valve cusps, provided no regurgitation is present, are thickened, though capable of closing the orifice. This sign must generally be regarded as unfavorable and the evidences of coronary obstruction may be looked for, especially if there is shortness of breath on exertion and præcordial dulness.

Diagnosis.—The presence of a systolic murmur at the second interspace to the right of the sternum, transmitted upward along the carotid arteries, is almost sufficient to establish a diagnosis of aortic obstruction. A small, slow pulse, hypertrophy of the left ventricle, the presence of a thrill over the base of the heart, are signs which will emphasize the diagnosis, although they are somewhat variable.

The conditions other than aortic obstruction which may be attended with the development of a basic systolic murmur and which, therefore, may prove a source of confusion in diagnosis, are roughening and rigidity of the valves, aneurysm, anæmia, obstruction of the pulmonary orifice, pressure of intra-thoracic tumors upon the aorta, patent ductus arteriosus and acute aortitis.

Roughening or rigidity of the valves may, like aortic obstruction, appear or be first discovered after middle age, and like it also may be attended with a systolic murmur over the aortic valve. It may be distinguished from aortic obstruction mainly by the accentuation of the second aortic sound which is unduly intensified on account of the high arterial tension. Illustrations may frequently be noticed in cases of gout, interstitial nephritis and other disorders of advancing life accompanied by high arterial tension and the slow development of a murmur over the aorta.

Aneurysm of the ascending aorta may sometimes be mistaken for aortic obstruction. This can only happen when the dilatation is fusiform in shape as the sacculated variety is not likely to lead to difficulty on account of its characteristic physical signs. There is in fact a wider channel through which the blood is propelled in comparison to which the orifice is constricted; thus in one sense there is a stenosis and the murmur is transmuted upwards along the vessel. The features by which simple fusiform aneurysmal dilatation may be known are a greater extent of area of dulness, outward to the right at the line of the second and third costal cartilages (this sign is sometimes absent), the presence of a soft, blowing systolic murmur (produced by the eddies which occur in the dilated aorta) and accentuation of the second aortic sound, caused by the greater column of blood falling back on the cusps. Aneurysm is sometimes also attended with inequalities of the pulse on one or other side, while in aortic stenosis the pulse is the same on both sides, being small and hard and almost always slow. Palpitation, breathlessness, vertigo and pain are less prominent in aneurysm. For other signs of aneurysm see that subject.

The anæmic murmur which is heard corresponding in location and rhythm with that of aortic obstruction is distinguished by the following features: Feebleness of impulse, soft, full, compressible pulse, absence of hypertrophy, greatest intensity of murmur over carotids, lack of uniformity in direction of conduction, and the presence of a more or less marked venous hum. In contrast, in aortic obstruction there is increased force of the impulse beats, the pulse is slow, small and full, there is hypertrophy of the left ventricle, and the seat of the maximum intensity of the murmur is at the junction of the second rib with the sternum, while the direction of conduction is upwards along the carotids. The dyspnœa in anæmia shows an additional point of difference in that it is painless and panting, while that of aortic obstruction partakes of a sense of oppression which may amount almost to pain. The aortic area is the most infrequent locality in which anæmic or neuro-cardiac murmurs appear.

When the aortic systolic murmur is heard with its greatest intensity at the left of the sternum, as has been mentioned it sometimes may, it may be confounded with pulmonic obstruction. A distinction may be made by recalling the fact that the pulmonic murmur is never propagated to the cervical vessels. The extreme rarity of the lesion should also be borne in mind.

Pressure upon the aorta of tumors within the thorax may give rise to a basic systolic murmur. These growths may arise from the remains of the thymus gland, from the mediastinal connective tissue, from the parietal pericardium and from the periosteum of the bones of the thorax. It is needless to say that these conditions are not common, growths on the pericardial surface and bony structure of the thorax being extremely rare. The nature of these neoplasms are usually of the small-celled type. They may make their appearance in connection with leucocythæmia and lymphadenoma; others may be tubercular, syphilitic or malignant. When a tumor presses upon the aorta so as to cause constriction, in some instances pulsation of the vessel may develop which may closely simulate aneurysm, but in others there may be a systolic murmur heard over the upper part of the sternum transmitted upward along the great vessels of the neck. The points of difference by which the latter condition may be distinguished from aortic obstruction are the extent and variability of the area of dulness on percussion which accompanies morbid growths, and which varies accordingly, the absence of structural changes in the arteries and changes in the second sound and the history of the case, the latter, in many instances, will assist diagnosis.

Acute aortitis, a rare and obscure affection, may be attended with a basic systolic murmur. It is recognized by the presence of irregular pyrexia, rapid pulse, dull pain and tendency to heart failure.

Patent ductus arteriosus, as stated when considering congenital affections of the heart, is attended with a systolic murmur heard with the greatest intensity in the second left intercostal space, an inch and a half from the sternum. The fact that the murmur is not transmitted to the vessels of the neck and that it is late systolic in rhythm distinguishes it from aortic obstruction.

Prognosis.—Aortic obstruction is generally regarded by many as the least unfavorable of cardiac lesions. When the stenosis exists in only a moderate degree compensation is readily established and may continue for a considerable period. This favorable aspect of the condition, however, is greatly modified by the fact that the pathological changes may affect the coronary circulation. Under these circumstances there may be distressing symptoms and sudden death. It may be said, however, that notwithstanding its relation to arterio-sclerosis as a causative factor the outlook is decidedly more favorable than in aortic insufficiency, and there is no risk of fatal syncope so long as symptoms of arterio-sclerosis are absent. When, however, compensation has broken down and dropsy appears, there is less chance of relieving the condition than in other valvular affections.

When the lesion arises from endocarditis in a young person compensation is easily established and maintained, but the possibility of the progress of the endocardial inflammatory processes is an element of danger which at some time may lead to the development of symptoms. A systolic murmur appearing in middle or advanced life, as far as the obstruction in the aortic orifice is concerned, may not be regarded of itself as of such serious import, but, on the other hand, as an indication of degenerative changes it must be looked upon as of serious significance.

Treatment.—Aortic obstruction scarcely calls for special details of treatment. The remedial measures recommended for insufficiency of the aortic valves are here applicable. When a gouty diathesis is the underlying cause the diet should be regulated accordingly. The heart should be spared unnecessary muscular effort or emotional disturbance. Rest will at times be indicated.

The limitation of the arterio-sclerosis by the use of the iodides, as in aortic insufficiency, may be attempted. Palpitation and angina should be met on the same line of treatment as elsewhere mentioned. Care should be taken against contracting pulmonary affections.

Tricuspid Insufficiency.

Insufficiency of the tricuspid valve is in complete closure of the valve ; the blood in consequence regurgitates through the tricuspid orifice from the right ventricle into the right auricle during systole. Formerly the lesion was looked upon as rare, but now it is regarded as frequent, although as a primary affection it is very uncommon.

Under certain conditions tricuspid regurgitation may be considered as physiological. This occurs when the right ventricle becomes over-distended by violent exertion, whereupon a reflux of blood follows by which the intracardiac pressure of the right ventricle is relieved. In many text-books this phenomenon is described as the safety-valve action of the tricuspid. As a rule there is no murmur.

Ætiology.—Incompetency of the tricuspid valve may be produced by endocarditis, degeneration of the valve and the effects of changes in the muscular walls of the heart.

Lesions arising from endocarditis are rare and are confined for the most part to intrauterine life. This is due to the fact that the fœtal circulation puts more work on the tricuspid valve, while it leaves the mitral valve comparatively free. Bramwell, however, has pointed out that tricuspid insufficiency occurs in young children and is more frequent than was formerly supposed. Other rare instances of primary occurrence may be found in association with ulcerative endocarditis from septicæmia, gonorrhœa and pneumonia.

Degeneration of the valve segments is not an infrequent cause, but rarely is the process seated primarily in the tricuspid. When it occurs it is generally in association with obstruction, arising secondarily to valvular deficiencies of the left side of the heart, more especially those of the mitral orifice, or it may form a part of a widespread degenerative process.

Changes in the heart walls constitute the most fruitful source of tricuspid insufficiency. The casual factors are many and include all these influences which tend to induce increased ventricular tension with consequent ultimate dilatation and atheroma. These conditions are for the most part the secondary effects of mitral lesions, chronic bronchitis, emphysema, fibroid changes in the lungs, the later stages of chronic interstitial nephritis and lesions of the pulmonary valve. Influences acting directly upon the heart walls in general necessarily affect the right ventricle. These conditions include pyrexia, toxæmia and malnutrition, and as a rule are not permanent in their effects.

Both mitral insufficiency and obstruction lead to stagnation of the pulmonary circulation. In both instances the blood accumulates first in the left auricle, then follows damming back of the circulation in the pulmonary veins, lungs and pulmonary artery. The obvious effect of this chain of consequences is increased blood-pressure in the right ventricle. The natural result is dilatation and hypertrophy. For a time these changes enable the ventricle to fully compensate, but the increased tension ultimately leads to degeneration in the valve structure and myocardium and deficiency follows. Aortic disease in the absence of mitral lesions has no effect upon the right side of the heart.

Chronic bronchitis when it becomes sufficiently protracted may develop dilatation of the bronchial tubes and an emphysematous condition of the lungs. In consequence the pulmonary circulation suffers from obstruction, the effect of which falls upon the right ventricle causing dilatation and tricuspid insufficiency. Fibroid phthisis or pulmonary cirrhosis may be attended with the same result.

Chronic interstitial nephritis when it has reached the stage of obstruction of the renal circulation with increased tension of the left ventricle, unless other complications terminate the case, leads to dilatation and hypertrophy with the sequence of cardiac and pulmonary circulatory changes as mentioned.

In association with these changes there may be not only dilatation of the heart walls, but also more or less degeneration, due to defective nutrition and disease of the coronary arteries which contribute to the causation of the valvular deficiency.

Obstruction of the pulmonary orifice has a most decided effect in causing dilatation of the right ventricle with consequent changes in its walls. It is a most direct factor in producing tricuspid insufficiency, but the rare occurrence of this lesion renders it one of the least important in the ætiology of tricuspid disease.

Pyrexia if at all continued may be attended with a relaxation of the muscular wall of the right ventricle and, in some instances, by a hyaline degeneration of its muscular structures. Toxic conditions of the blood, namely, the presence of micro-organisms, the mineral poisons and alcohol produce the same effect. Malnutrition from any cause may also be attended with the same phenomenon.

Morbid Anatomy.—The pathological changes which result from endocarditis and degeneration follow the same course as in the instance of the mitral valve. In cases of endocardial inflammatory origin the structure of the cusps undergo thickening and induration which is subsequently followed by contraction and adhesions with implication of the chordæ tendineæ. In another and more unusual type the surface of the valve is covered with vegetations and cauliflower-like excrescences. Degenerative lesions likewise produce thickening, rigidity and distortion of the valve segments. In some instances the morbid processes is more active in the columnæ carneæ which lose their mobility. In exceptional cases the valve undergoes ulceration or may be the seat of abscess or aneurysm. Perforation under these conditions may sometimes occur.

In the majority of cases there is no direct lesion of the valves.

The changes are confined to the walls of the ventricle and consist of dilatation leading to stretching of the ventricular orifice and consequent incomplete co-adaptation of the valve cusps. Under these circumstances the cusps may remain intact.

MECHANICAL EFFECTS OF THE LESION.—With each systole of the right ventricle there is regurgitation of the blood into the right auricle which thus becomes distended and dilated. In some cases there is hypertrophy, while in others dilatation accompanied by marked thinning of the auricular walls. The result of the accumulation of blood in the right auricle obviously leads to retardation of the flow from the superior and inferior vena cava which become more or less dilated. There now follows pulsation of the jugulars and more or less stasis generally of the venous circulation with the concomitant symptoms of dropsy, gastro-intestinal catarrh and hepatic engorgement as described under mitral insufficiency.

A necessary consequence of the reflux of blood through the tricuspid orifice is a diminution of the amount supplied to the pulmonary arteries, notwithstanding the fact that these vessels may be in a condition of engorgement.

For a time the right ventricle maintains the equilibrium of the pulmonary circulation, but soon fails and undergoes dilatation with thinning of its walls. In some instances the dilatation is extreme.

Symptoms.—Mild degrees of insufficiency may exist without exciting attention, but when the valvular defect is marked the symptoms are prominent. In most cases the primary affection of the heart is overshadowed by the symptoms attending tricuspid incompetency.

An early symptom is swelling of the ankle. In slight cases this may only be present at night, disappearing in the morning after rest. Various gastric and intestinal disorders appear; anorexia, indigestion, diarrhœa, or constipation, and a sensation of weight in the region of the liver.

Cyanosis is prominent, especially in advanced cases. In some instances it is associated with a slight jaundiced hue, bordering rather on a greenish than a yellow tinge.

Except in those rare cases where the disease arises primarily in the right ventricle, the respiration shows marked disturbance. The difficult respiration is generally more of the form of orthopnœa.

The breathing may be short and panting and paroxysms of dyspnœa are excited without apparent cause. As the case advances the respiration becomes more impeded. The patient will be unable to recline and will be compelled to sit continuously upright, seeking relief by sitting near an open window or by being constantly fanned. In some instances the dyspnœa is not so constant, and there may be intervals of respite, but paroxysms occur attended with intense suffering. The condition is that of cardiac asthma from spasm of the bronchial vessels arising from weakness of the heart. During these attacks every effort aggravates, the skin is bathed in a cold perspiration, the patient struggles to inflate the lungs, the expression of the face denotes great suffering and the vessels of the neck throb. Sometimes the paroxysms arouse the patient from sleep. The heart action at first is generally tumultuous, but later becomes weak, and with the subsidence of the paroxysm the weakness becomes intensified. The pulse is arrhythmic and almost imperceptible. Œdema of the lungs is liable to ensue which in many instances leads to a fatal termination. Pulmonary œdema is also a frequent complication of tricuspid insufficiency, apart from cardiac asthma, more especially as a sequela of mitral insufficiency.

Hydrothorax is more prone to occur with disease of the right side of the heart than with the left. This is due to the fact that the blood of the pleural and pulmonary circulation for the most part flows back to the heart through the bronchial veins which, on the right side, discharge by means of the vena azygos major into the vena cava superior, and on the left by means of the superior intercostal veins into the venæ innominatæ. Thus the course of the blood is to the right auricle. When, therefore, the right auricle is in a state of distention from a right side lesion, there arises a backward pressure upon the pleural circulation which leads to the effusion into the pleural cavity. In the same way, tricuspid insufficiency, by impeding the bronchial veins, leads to catarrhal conditions in the lungs. The presence of hydrothorax is not infrequently the cause of persistent forms of dyspnœa or the source of aggravation of this distressing condition.

Dropsy is characteristic of tricuspid incompetence. When constant it is a sign that the lesion is advanced. It may and generally does begin at the ankles and usually ascends until the patient is more or less "water-logged." It is needless to dwell upon the mechanism of the circulatory disturbances which lead to the development of general anasarca, as that subject has been discussed in connection with mitral disease. In most cases the liver is enlarged, congested, tender and painful. The pain is due to the tension of the capsule from swelling of the organ. The kidneys also show marked signs of disturbance. The secretion of urine is generally less than normal with decrease in the amount of urea, deeper color and increased specific gravity. Nephritic complications may become subsequently more serious, as evidenced by the presence of casts and albumin.

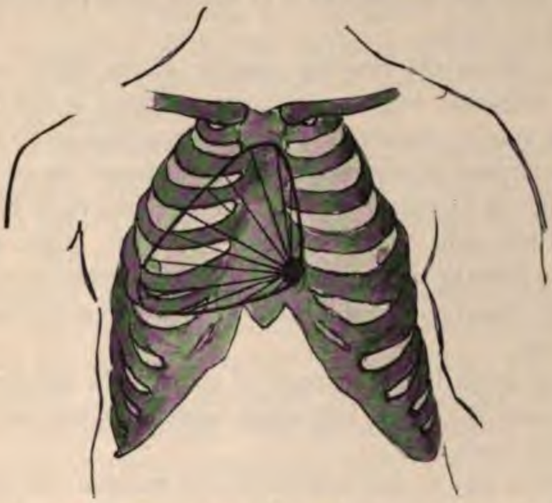

FIG. 27.—Area of the tricuspid systolic murmur, black circle indicates the point of maximum intensity, the lines show the possible direction of transmission.

Nervous symptoms are frequent. Headache is often troublesome and sleep disturbed. Sometimes the patient is harassed by distressing dreams which cause him to awaken suddenly. In some cases sleep may be possible during the day and quite impossible at night. Mental disturbances are not uncommon; illusions and hallucinations may appear. The pulse is not characteristic; it is usually small in volume and of diminished pressure.

PHYSICAL SIGNS.—*Inspection* reveals the presence of pulsation of the jugular veins caused by the backward pulse wave of the right ventricular systole. This sign is pathognomonic of tricuspid insufficiency. It may be observed to the best advantage by placing the patient in the semi-recumbent position. It is more marked on the right side than on the left. In exceptional instances the subclavian and axillary veins may also be seen to pulsate. In many cases the superficial veins stand out showing marked turgescence. Epigastric pulsation is common. It is due to the movements of the enlarged right ventricle. Occasionally, there may be pulsation in the third and fourth intercostal interspaces very near the sternum; these arise from the regurgitation of blood from the enlarged ventricle into the auricle.

Palpation shows the heaving impulse of the right ventricle in the upper epigastric region. Hepatic pulsation is also frequently present and may be detected to the best advantage when the patient is in the recumbent posture with the arms raised above the head. The examiner should place the left hand over the right mid-axillary region and the right over the upper abdomen. Care should be taken not to mistake the impulse of the right ventricle transmitted through the diaphragm to the left lobe of the liver for that of hepatic pulsation.

Percussion gives lateral increase of the area of dulness more especially toward the right, extending sometimes beyond the right border of the sternum.

Auscultation may or may not discover a murmur, for its presence in tricuspid insufficiency is by no means constant. In a certain proportion of cases none can be heard. This occurs more especially in functional regurgitation. Even when the "safety-valve" action has developed marked valvular deficiency, as shown by the presence of jugular pulsation, there may be no appreciable murmur. In such cases it is common to find the first sound in the tricuspid area weak. In many cases, however, an adventitious systolic sound is heard, with its greatest intensity at the junction of the fifth and sixth left costal cartilages with the sternum. Some observers give the base of the ensiform cartilage as the point where this murmur is most clearly audible. It is transmitted from its point of greatest intensity in all directions and frequently it is difficult to separate it from aortic and pulmonic murmurs. Its quality is soft and blowing.

The second pulmonic sound is generally enfeebled, but is subject to variations in intensity, and may sometimes be intensified. For example, when the pressure in the pulmonic circuit is relatively increased and remains so, while that of the general circulation is lessened, either accentuation or doubling of the second pulmonic sound may be heard.

Diagnosis.—The salient points of diagnosis are the presence of a systolic epigastric pulsation, venous pulsation visible in the neck, hepatic pulsation recognizable by palpation, and a feeble second pulmonary sound. A systolic murmur heard with its greatest intensity at the junction of the fifth and sixth ribs to the right of the sternum is important additional evidence, but venous pulsation alone is sufficient to establish the diagnosis.

In cases of mild types the only indication pointing to the presence of tricuspid insufficiency may be that of the murmur, but inasmuch as a systolic murmur may be transmitted to the tricuspid area from other parts of the heart, error is possible. Confusion may be avoided by distinguishing the location of the maximum intensity of the murmur in question. It is scarcely possible for a pronounced tricuspid insufficiency to be confused with any other heart lesion, for its pathognomonic sign of venous pulsation will remove all element of doubt. The murmur, however, is frequently difficult to detect when in association with, or secondary to, mitral insufficiency.

Prognosis.—In slight forms of tricuspid insufficiency the outlook is favorable. In functional types and those arising from pyrexia, toxæmia and temporary disturbance of the lungs, the prognosis is decidedly favorable. It is far otherwise, however, when the lesion occurs in consequence of disease of the left side of the heart. Here its appearance must generally be looked upon as the precursor of an ultimately fatal issue.

Treatment.—The general treatment suggested for valvular lesions will be indicated, modified according to the condition with which the lesion is secondarily associated. It will, therefore, be more or less varied as to whether it has arisen in association with disease of the mitral valve, pulmonary affections, faulty nutrition with gastric catarrh and dilatation of the stomach, or whether it simply arises from the relaxing influences of pyrexia or toxæmia. In all cases remedies of the cardiac tonic class will be found called for, among which digitalis stands first. Conditions of threatened collapse, of which paroxysms of cardiac asthma or pulmonary œdema are premonitory, will demand the prompt administration of stimulants, such as caffein or strychnia, hypodermically. Frequently, digitalis affords speedy relief.

Tricuspid Obstruction.

Tricuspid obstruction is an impediment to the blood as it flows from the right auricle into the right ventricle, during diastole. The lesion is very rare. Its independent existence is exceptional. An overwhelming proportion of cases occur in association with other valvular deficiencies. Congenital cases, which form a large percentage of those on record, are invariably accompanied by some other form of valvular anomaly, while antenatal cases are most always associated with tricuspid insufficiency or mitral stenosis.

Ætiology.—Abnormal fœtal development is, according to some observers, the most frequent source of the lesion, but the views of the majority of writers point to an antenatal origin for the larger proportion of cases.

Among adults it is very frequently preceded by mitral stenosis, and in the majority of instances is generally attributable to acute rheumatism, which is responsible for about one-half of the cases on record. Others have been traced to acute specific diseases ; some, again, have been found associated with chorea, while in not a few the origin cannot be traced.

Statistics show that the lesion occurs with greater frequency amongst females than males. There does not appear to be any satisfactory explanation for this characteristic.

Morbid Anatomy.—The anatomical changes follow the same process as when seated in the mitral orifice. In some of the cases reported the stricture was found to have arisen from adhesions between the cusps resulting in a funnel-shaped mass which surrounded the orifice and protruded into the right ventricle. The degree of contraction in lesions of this nature may be excessive. It may be so great as to scarcely admit a finger. Associated with the adhesive process there may be more or less induration and rigidity.

Structural alterations of the vegetative type also appear. They may present a variety of pathological changes similar to that which occurs when seated in the left auriculo-ventricular orifice.

When tricuspid obstruction is congenital it is usually accompanied by other malformations, such as pulmonary stenosis, patent foramen ovale or patent ductus arteriosus.

Mechanical Effects of the Lesion.—Tricuspid obstruction leads at once to the accumulation of blood in the right auricle, which dilates and expands sometimes to an excessive degree. The auricle, however, is poorly adapted to compensation and accomplishes little in that respect. The presence of the excess of blood in it tends to the accumulation of blood in the great veins, and ultimate retardation of the venous circulation with general systemic stasis.

Symptoms.—The symptomatology of tricuspid obstruction in most instances is overshadowed by that of the lesion which it accompanies. This fact, together with the infrequency of its separate existence, renders the clinical course more or less obscure. The symptoms which point to its presence are dyspnœa, cyanosis, hepatic enlargement, renal complications and a marked tendency to a general stasis of the venous system. These symptoms, on the other hand, are found in association with tricuspid insufficiency. It is therefore difficult to determine their relationship to the obstructive lesion. The presence of the latter generally tends to aggravate, especially the cyanosis, which is more marked than in insufficiency. There is less duskiness of the countenance and sometimes an icteric hue. Paroxysms of cardiac asthma are frequent, while dyspnœa is more or less continuous and persistent if the obstruction is at all considerable. There is ascites, œdema of the lower extremities, headache, hebetude and marked mental disturbances. The urine may be scanty and albuminous. When it can be recognized the physical signs are as follows:

Physical Signs.—*Inspection* shows turgescence of the veins of the neck which may pulsate feebly or remain motionless.

Palpation may detect a presystolic thrill with a variable area of maximum intensity, which may sometimes be felt at the apex of the heart at the fifth interspace, sometimes outside the left edge of the sternum.

Percussion may give lateral increase of the area of dulness.

Auscultation discloses a presystolic or diastolic murmur in a certain proportion of cases; according to G. Newton Pitt, in about one-eighth. Its point of greatest intensity is at the junction of the fifth or sixth interspace on the left of the sternum. An epigastric bruit is common.

These physical signs are those of uncomplicated cases, which, as has been stated, are rarely seen.

Diagnosis.—If a presystolic murmur is recognized with its point of maximum intensity in the tricuspid area, it may be regarded as pathognomonic, but taking into consideration the fact that the murmur is more frequently absent than present, and that when present it is most always associated with other adventitious heart-sounds, it is needless to say that the diagnosis is often impossible and in many cases the lesion is not recognized until revealed by post-mortem examination.

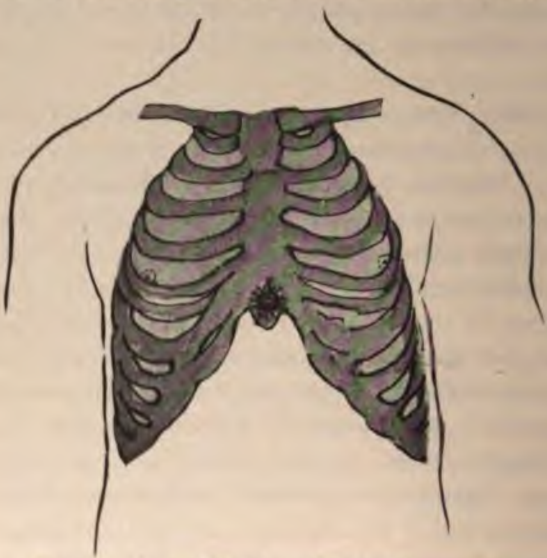

FIG. 28.—Area of the tricuspid presystolic murmur.

Prognosis.—The prognosis is unfavorable. The forecast of the future is necessarily greatly influenced by the presence of associated lesions. It will be thus necessary to consider each case separately.

Treatment.—Treatment does not call for individualization, as it is obviously that of mitral disease, especially stenosis and tricuspid insufficiency. Remedial measures, however, are here less likely to prove beneficial.

Pulmonary Insufficiency.

Insufficiency of the pulmonary valve implies imperfect closure of the valvular segments and reflux of blood during diastole through the pulmonary orifice into the right ventricle. It is the rarest of valvular lesions. According to Guy's Hospital Reports, extending over a period of twenty-four years, it was found in only seventeen cases out of eleven thousand.

Ætiology.—A considerable number of the recorded cases are congenital in association with other deformities of the heart structures. Non-congenital cases arise from the effects of endocarditis, rheumatism, ulcerative degeneration, dilatation of the pulmonary artery, pulmonary obstruction and the pressure of aortic aneurysm. In some instances no apparent cause can be found.

Morbid Anatomy.—Lesions of endocardial or degenerative origin follow the same general line of structural alteration as when seated in the aortic area; thickening, induration, contraction, adhesion and deformity with occasional ulceration and perforation. Atheroma or arterio-sclerosis involving the pulmonic valve causing insufficiency has been traced in several instances to syphilis. Arterio-sclerotic processes when seated in the pulmonic orifice may, by contraction, or other obstructive conditions, interfere with the action of the valve and thus permit leakage.

Dilatation of the pulmonary artery may also give rise to regurgitation. Mechanical pressure from an aortic aneurysm on the valve may likewise interfere with its action and form a source of incompetency. It is well to remember that the valves on the right side of the heart are less perfect in action than those of the left (Hunter), and a certain amount of relative or functional incompetence may exist. Sex does not appear to influence the occurrence of the lesion.

MECHANICAL EFFECTS OF THE LESION.—The result of the leakage of the pulmonic valve is accumulation of blood in the right ventricle and increased tension; hypertrophy develops which for a time may compensate. All the strain, however, is upon the right ventricle, which, being compelled to bear it alone, soon gives way, whereupon tricuspid insufficiency follows. As soon as this lesion is established there follows the chain of profound functional and structural disturbances attendant upon general systemic venous stasis, which characterizes failure of the right side of the heart.

Symptoms.—While some of the text-books dwell to some extent upon the clinical course of pulmonic insufficiency, it must be said that practically its symptomatology is largely presumptive. This cannot be otherwise when taking into consideration the limited number of opportunities for observation, and the almost invariable presence of other valvular lesions in connection with it.

While it is thus difficult to describe the symptoms with any degree of accuracy, or to estimate their effects, the presence of pulmonary insufficiency cannot fail to be attended with the evidences of grave cardiac disease. Cyanosis and dyspnœa appear early, being at first occasional and excited for the most part only by exertion, but later both conditions are more or less constant. Clubbing and arching of the fingernails is a common symptom and as the lesion advances malnutrition becomes apparent and pulmonary affections may arise.

Pulmonary complications, especially in the form of hæmoptysis and catarrh, appear earlier from the fact that the compensating hypertrophy of the right ventricle gives way more readily.

The pulse does not present any special characteristics. It may be slow and irregular and indicative of low pressure.

PHYSICAL SIGNS.—The physical signs depend chiefly upon dilatation and hypertrophy and the evidences afforded by auscultation. They are not, however, clear or distinctive. *Inspection* may show distention of the veins of the neck, which sometimes pulsates. The apex-beat of the heart may be observed to be displaced to the left beyond the mammillary line. There may be marked epigastric pulsation, and pulsation in the intercostal spaces to the right of the sternum.

Palpation may discover a diastolic vibratory thrill with the second sound and confirm the signs elicited by auscultation.

Percussion may show increase in the area of dulness to the right, extending to and beyond the right margin of the sternum when the hypertrophy of the right ventricle is extreme.

Auscultation may furnish a diffuse diastolic bruit audible over a wide area, but with the point of its maximum intensity in the left second intercostal space. It may be transmitted downward along the left border of the sternum, but is not heard at the apex. It varies in character, being sometimes blowing and sometimes harsh. Generally it is loud, displacing the second sound entirely. When associated with a similar murmur of aortic origin it may exceed the latter in intensity.

Diagnosis.—The recognition of the lesion depends upon the presence of a diastolic murmur and thrill in the second intercostal space to the left of the sternum, increase of the area of dulness to the right and marked cyanosis with more or less dyspnœa.

In congenital cases the presence of other anomalies of the heart structure must be taken into consideration.

Pulmonary insufficiency may be confounded with insufficiency of the aortic valve and aneurysm of the aorta.

Aortic insufficiency is the most probable source of error, especially as the aortic murmur may sometimes be found on the left side of the sternum instead of its usual location on the right. To distinguish between the two lesions the following characteristics of aortic insufficiency should be recalled to mind: namely, its murmur is transmitted directly toward the apex where it is distinctly audible, it is heard over the carotid artery, and accompanied by the Corrigan's or water-hammer pulse and capillary pulsation. On the other hand, pulmonic insufficiency is not transmitted directly to the apex, it may be transmitted downward along the left border of the sternum, but is not heard over the apex, it likewise is inaudible over the carotids and is never attended with the water-hammer pulse or capillary pulsation. It is, on the other hand, accompanied by cyanosis, dyspnœa and clubbing of the fingernails, conditions, however, which may attend heart failure from any lesion.

Aneurysm of the aorta may be distinguished from pulmonic insufficiency by the fact that its bruit is systolic instead of diastolic, by the irregularity of its murmurs which do not coincide exactly with the heart action and by the variety of the localities in which its maximum intensity may be located. It is further characterized by a more extensive area of dulness which is not confined to the region over the right ventricle. There may be also pressure symptoms attending aneurysm which are never present in association with pulmonic insufficiency.

Prognosis.—The future in cases of pulmonic insufficiency is grave. The tendency to disturbance of the pulmonary circulation and development of pulmonary complications which place additional stress upon the right side of the heart are especially unfavorable. Yet, notwithstanding the strain upon it, the right ventricle may compensate in some instances for a considerable period.

Acute inflammation of the lungs and bronchial tubes, whooping-cough and measles is attended with the greatest danger in children with pulmonic insufficiency. Frerichs regards the lesion as favorable to the development of tuberculosis.

Treatment.—The tendency to affection of the lungs and bronchial tubes and disturbances of the pulmonary circulation should be met with special preventive measures. Care in regard to exposure, deficient ventilation, and the vicissitudes of climate should be carefully enjoined. As far as the care of the lesion is concerned, beyond particular attention to the lungs, the general deductions will be found to cover all indications. In the case of children, prophylaxis of infectious diseases, it is needless to say, is a matter of much importance.

Pulmonic Obstruction.

Pulmonic obstruction is an impediment to the blood as it flows from the right ventricle into the pulmonary arteries during systole. It is the most frequent congenital cardiac defect and one of the rarest of acquired lesions.

Ætiology.—In cases which develop after birth the same general causes are operative as in other valvular affections. Of these, endocarditis is the most active factor. With a few exceptions, the endocardial inflammation is primarily seated in some other valve from which it extends to the pulmonic. Von Wahl reported a case in which it was confined to the pulmonic valve. Of the infectious diseases which give rise to endocarditis, rheumatism, while accountable for some cases, is probably less prominently connected with the causation of pulmonic stenosis than it is with other forms of valvular disease. Schwalbe reports cicatricial contraction preceding stenosis of the pulmonic orifice as due to syphilis. Another source of pulmonary obstruction is endarteritis of the pulmonary artery, which has been found in rare instances.

Among cases from isolated causes which cannot be classified are neoplasmata of neighboring organs which press upon the valve structure, direct injury and gummatous infiltration of the pulmonary artery. In some instances no assignable cause can be recognized.

Morbid Anatomy.—According to Vimont, the changes are confined to the cusps in the greater proportion of cases (twenty-two out of thirty-two).* The valve segments are usually found adherent along their margins, the union producing either a sort of diaphragm or a cone-shaped mass which projects into the pulmonary artery. Cases of the latter type are mostly of congenital origin. The structural alterations consist, as in other conditions, of thickening, induration, rigidity and calcification and are attended with incompetence as well as obstruction.

In association with this condition, vegetations may be found on the cusps, or their presence alone may constitute their sole feature, forming a marked degree of obstruction.

In some cases the pathological process may be seated beyond the valve in the pulmonary artery, when it will depend on the presence of arterio-sclerosis or atheroma.

Paul describes a form of obstruction which arises in the cornus arteriosus, due to ventricular endocarditis, which involves the cusps by extension, and which is associated in some instances with a communication between the right and left ventricle, caused by actual perforation of the ventricular septum from myocarditis.

MECHANICAL EFFECTS OF THE LESION.—The resistance of the obstacle at the pulmonic orifice causes excess of blood in the right ventricle, which accordingly hypertrophies, sometimes becoming thicker than the left. Similar changes in the right auricle result, except as to the degree of hypertrophy. Relative insufficiency of the tricuspid valve, with systemic stasis, in turn follows.

Symptoms.—In congenital obstruction cyanosis is marked, while in acquired cases this does not seem to be the case. Dyspnœa is usually absent, as the ventricular hypertrophy is able to compensate for a time and thus relieve the pulmonary circulation. Venous stasis and œdema are also absent, or if present are only manifest in a mild degree. Clubbing and arching of the nails is generally regarded as a marked symptom. A tendency to pulmonary phthisis is a characteristic of pulmonic obstruction. This is explained as due to a defective blood-supply. When compensation fails the symptoms of general venous stasis follow.

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* Twentieth Century Practice, New York, 1895, p. 287.

In congenital cases the associated anomalies of the heart may for a time compensate by relieving the right ventricle of the increased tension put upon it, but in these cases there is frequently arrest of the general development, mental as well as physical. The countenance is apt to be peculiar, the eyes are unduly prominent and the lips thickened, while the veins of the neck and head are frequently turgescent. The thorax is sometimes contracted, the præcordial region prominent and the abdomen protuberant. Paroxysms of breathlessness, hæmorrhage from the lungs or nose and convulsions may occur; mental torpor and insomnia are common. Dropsical symptoms are not usually marked.

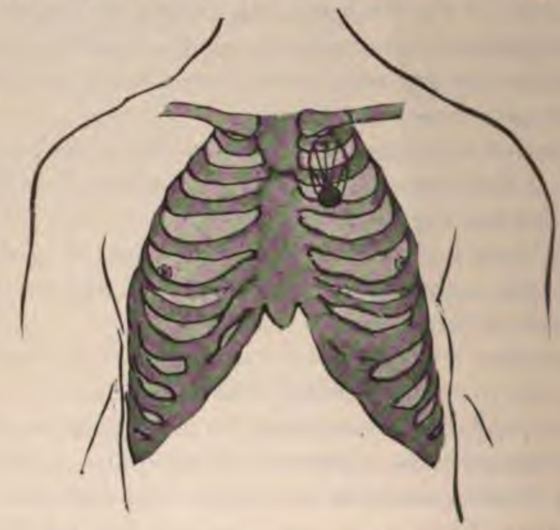

FIG. 29.—Area of the pulmonic systolic murmur. The dark circle indicates the point of maximum intensity, the elliptical shaded area, the limits of audibility, the arrow the direction and limit of transmission upward.

PHYSICAL SIGNS.—*Inspection*, as a rule, is negative, except in congenital cases, as mentioned. *Palpation* shows a systolic murmur and thrill over the base of the heart, extending in some instances over the præcordium. *Percussion* may disclose some enlargement of the normal area of dulness. *Auscultation* furnishes important evidence in the presence of a systolic murmur over the base of the heart, with its maximum intensity in the left second intercostal space, near the sternum. The systolic character is decided. In some instances the sound is prolonged to the second sound. A very important feature is that while considerably diffused, it is never heard over the great arteries of the neck.

The murmur is described generally as harsh and very loud. Nearness to the surface is given, by some observers, as a feature. Paul states that it is louder while the patient is in the recumbent posture than while standing or sitting. On the other hand, it is fainter when the lips and nose are closed and respiration is restrained.

Diagnosis.—There appears to be only one feature in non-congenital pulmonic obstruction which can be said to be clearly distinctive, that is, the existence of a systolic murmur over the base of the heart which is not transmitted upward along the great vessels of the neck. The diagnosis depends upon the recognition of this sign, as others, such as the point of maximum intensity, changes in the heart chambers and subjective symptoms are not sufficiently characteristic to be conclusive.

In congenital cases the diagnosis is usually not difficult. The consideration of this subject properly belongs under the head of congenital malformations.

The lesions which may form a source of confusion in diagnosis are aortic obstruction and patent ductus arteriosus. In the case of aortic obstruction there is often great difficulty, especially as the murmur of the aortic lesion is sometimes heard more distinctly over the left of the sternum. The great point of distinction lies in the fact that the pulmonic murmur is never propagated upwards to the cervical vessels, while that of aortic obstruction always is.

Patent ductus arteriosus may not only resemble pulmonic obstruction, but may also be associated with it. The presence of a point of maximum intensity at least two inches from the mid-sternum and its prolongation beyond the second sound indicates patent ductus arteriosus. Both these features are absent in pulmonic stenosis.

Prognosis.—Pulmonic obstruction is generally hopeless. Most cases die before adolescence. An exceptional few may arrive at maturity or old age. Death usually occurs from pulmonary œdema resulting from cardiac asthma, convulsions, pulmonary tuberculosis or catarrhal pneumonia.

Treatment.—The general methods will cover all that can be done. The bronchial and pulmonary affections which arise will obviously necessitate special measures for their relief. In conjunction with internal cardiac remedies, warm medicated inhalations will prove of assistance.

Associated Valvular Lesions.

The fact that a lesion affecting a valve seldom results in a single defect, but is usually at least twofold in its result, lends a very practical aspect to the association of valvular defects. These cöexistent affections when situated in the same valve are almost always the result of the same pathological process. Insufficiency and stenosis are generally associated, although one or the other predominates. As a rule the more distinct the murmur of incompetency the fainter that of obstruction, and the reverse. When more than one area of the heart is implicated it is either as the result of repeated attacks of endocarditis, or from contiguity wherein the morbid process extends from one valve to another. Thus the aortic valve may become affected from the aortic side of the mitral, or atheroma may extend from the aortic to the mitral valve. Again, the structural alterations which arise in the heart muscle, and those produced by the effects of increased tension on the valves, may result in the same conditions. Thus mitral insufficiency leads to tricuspid insufficiency, and aortic or pulmonic insufficiency to dilatation and hypertrophy of both ventricles, and ultimately to relative mitral and tricuspid incompetence. The same results may follow aortic or pulmonic obstruction.

In some instances the cöincidence of insufficiency and obstruction at the same valvular orifice may be more beneficial than otherwise, as one lesion may compensate the other.

Relative insufficiency of the mitral valve followed by aortic insufficiency is in some cases attended with beneficial effects, as it prevents overdistention of the left ventricle and of the arterial system. On the other hand, mitral insufficiency when secondary to aortic stenosis is unfavorable. While the cöexistence of the two lesions is generally regarded as more serious than simple obstruction, it is less so than incompetence alone.

Mitral Insufficiency and Obstruction.

The presence of mitral insufficiency and obstruction in association is far more common than the existence of either singly. As a rule insufficiency predominates.

The ætiology and pathology of the dual lesion naturally partakes of the nature of both insufficiency and obstruction, and inasmuch as it is of greater frequency, it follows that the various genetic factors are more active in its causation than they are in either of the individual valvular defects.

Endocardial inflammation plays a more important part than degeneration, but frequently the onset of the combined lesion is insidious and obscure and sometimes it is impossible to trace it to a definite cause.

The anatomical changes are generally more grave than in either insufficiency or obstruction alone, the auricle is more intensely affected and undergoes decided dilatation and hypertrophy, sometimes the walls are very thin and there is dilatation without hypertrophy. It can be readily seen how the pathological processes which have been previously discussed would produce a twofold deficiency. The result of these changes in a general way may be found to consist of vegetations upon the surface of the valves which prevent complete adaptation of the segments over the orifice, and at the same time act as obstructions to the blood-current ; in contraction, shrinkage and thickening of the cusps and chordæ tendineæ with roughening of the valve surfaces, whereby the action of the valve is not only prevented, but the segments are caused to protrude as obstructions, frequently in a condition of fusion or blending, at the same time there may be infiltration around the orifice, which in turn causes narrowing of the same and loss of mobility of the segments.

The clinical course naturally shows the symptoms of both insufficiency and obstruction, but the equilibrium of compensation is often more readily unbalanced and grave disturbances appear earlier than in either single process.

Auscultation reveals a presystolic or diastolic murmur as well as a systolic sound; both are heard with the greatest intensity at the apex. In most instances the systolic murmur of insufficiency predominates to such a degree over the presystolic murmur as to render the latter difficult of detection. In these conditions the more rapid development of dilatation of the left auricle, the presence of decided accentuation of the second pulmonic sound, which is usually more marked in the double lesion, the irregularity of the heart action and the more decided development of dilatation and hypertrophy of the right ventricle points to the presence of a combined lesion.

The condition of the patient during the progress of the disease is attended with more distress and suffering than in any other combination of valvular deficiencies.

The prognosis is more grave than in either insufficiency or obstruction.

Treatment does not call for any specialization.

Aortic Insufficiency and Obstruction.

As in the case of the mitral orifice, aortic lesions are more generally encountered in association than singly. The pathology, ætiology and clinical course of the combined lesions necessarily consist of a blending of the conditions common to both, with a tendency to increased severity. The physical signs sometimes reveal the predominance of one murmur, sometimes another. The fact that both lesions may be present and only one recognizable is an important feature. This occurs more frequently in the instance of the diastolic or regurgitant murmur which may be absent in conditions of excessive stenosis, sometimes in association mitral disease. Again, a systolic murmur in the region of the aortic valve may be caused by changes in the walls of the ascending aorta and not by stenosis. The possibility of mistaking aneurysm of the arch of the aorta which is attended with a double murmur, of which the systolic portion is due to the roughness of the aorta and the walls of the aneurysm and the diastolic arises from the relative insufficiency due to dilatation of the aortic orifice, is an important consideration. The difference may be recognized by the fact that in aneurysm there is less hypertrophy of the heart than in aortic disease, the patient is usually older, there is no history of rheumatism and there are often other characteristic symptoms.

In treatment, as the obstruction is usually the less disturbing element, the conditions arising from incompetence will demand more attention.

Other valve lesions which are found in association with more or less frequency are aortic insufficiency and mitral obstruction, insufficiency of both mitral and aortic valves, mitral insufficiency and aortic obstruction, obstruction of both the mitral and aortic valves, tricuspid lesions with mitral insufficiency, or obstruction or with aortic lesions.

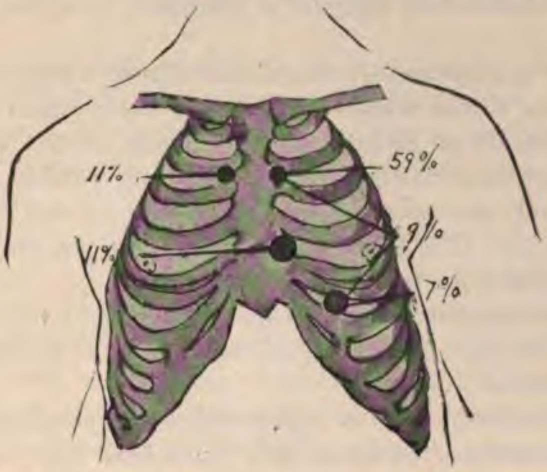

FIG. 30.—Percentage of the frequency of occurrence, according to Sansom, of anæmic murmurs as they occur at the various valvular areas.

It is scarcely necessary to discuss each of these conditions separately. It suffices to say that while the presence of one valvular defect necessarily modifies the clinical course and physical signs of other cöexisting lesions, the characteristic murmur of each must in most instances be recognized before a diagnosis would be warranted. The predominating lesion should first be determined, then the others together with the secondary structural changes in the heart which will correspond in most cases with the chief lesion. It is well to recall in this connection that mitral affections are more frequently secondary to aortic, and tricuspid to mitral.

ANEURYSM OF THE VALVES.

Aneurysm of the valves of the heart is a circumscribed sacculation on the surface of a valve segment. The lesion is essentially a part of some other pathological process and is not recognizable during life. Two forms are found: namely, that in which the whole thickness of the valve is affected and that in which one of the lamellæ has become ulcerated allowing the other to be pushed before it by the blood-pressure. The latter is usually the result of acute ulcerative endocarditis, although it sometimes is found in chronic conditions.

Valvular aneurysms vary in size from that of a pea to a pigeon's egg, and are almost always situated with their orifices toward the greatest amount of blood-pressure. Those on the aortic valve open toward the aorta and project into the left ventricle, while those on the mitral valve open toward the left ventricle and project into the left auricle. The mitral valve is their most frequent seat and it usually contains the larger varieties. (Foster.)

Valvular aneurysms vary in shape and number, but are generally round and single. The valves of the right side of the heart are seldom affected.

The usual termination is rupture followed by extensive insufficiency. Sometimes the aneurysm is found filled with coagula.

There are no definite symptoms indicative of the presence of valvular aneurysms and they may give no sign until rupture occurs. Aneurysm of the aortic valve, however, may cause a soft, systolic murmur which, on rupture, is attended with a murmur of insufficiency. When situated on the mitral valve they generally fail to give rise to any sign until rupture, when insufficiency suddenly appears.

SECTION IV.

DISEASES OF THE MYOCARDIUM.

MYOCARDITIS.

Myocarditis is inflammation of the heart muscle. It occurs in two forms, the acute and chronic, but these two types, in their origin, pathology and clinical course, have little in common.

Chronic myocarditis is considered under the title of Fibroid Degeneration.

Acute Myocarditis.

Acute myocarditis occurs in three varieties: the interstitial, the parenchymatous and the purulent (abscess of the myocardium).

Ætiology.—The disease is a secondary affection, and arises in connection with acute infectious and septic fevers, namely, acute rheumatism, scarlet fever, diphtheria, smallpox, pneumonia, erysipelas, malaria, cerebro-spinal meningitis, typhoid, pyæmia and septicæmia. In rare instances it has been found in association with gonorrhœa. Its occurrence, on the other hand, with the infectious fevers is not unusual. There is reason to believe that it may be caused by the severe form of influenza or la grippe.

When secondary to rheumatism it is the result of extension of the inflammatory processes of the endo- and pericardium to the heart muscle. In other forms it is caused by the toxic condition of the blood acting directly on the myocardium or by the deposit of infected emboli in its tissues.

Males are more prone to the affection than females.

Morbid Anatomy.—The pathological changes may be either general or localized; more commonly they are the latter, being confined, as a rule, to one point. The usual seat is apex of the left ventricle. During fœtal life the right ventricle appears to be the point affected. The auricles generally escape.

In the interstitial form which is more commonly observed in association with acute pericarditis and infectious diseases, the structural alterations show swelling and small, round cell and leucocyte infiltration of the intermuscular connective tissue. The bloodvessels may be dilated and the muscular fibres the seat of granular, fatty or hyaline degeneration. In the instance of pyæmia there may be infiltrated pus cells which are confined to the interstitial tissues. The more frequent examples of these changes are found in typhoid fever, acute endocarditis and diphtheria. In some instances the inflammation may be the starting-point of fibrous or chronic myocarditis.

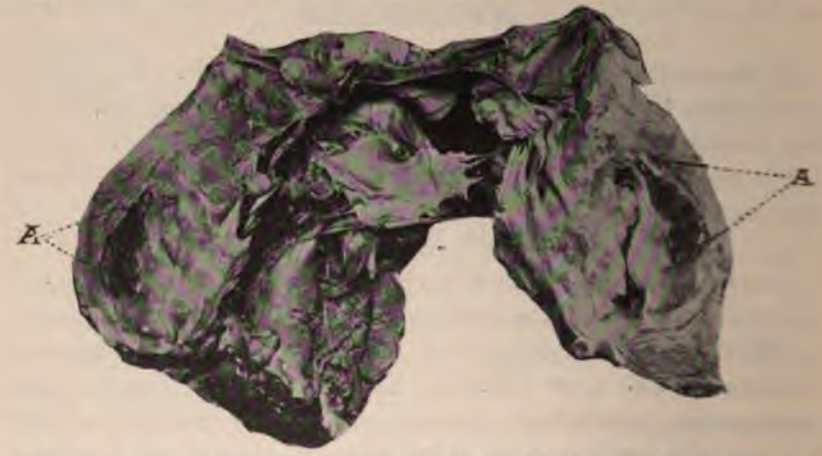

FIG. 31.—Acute suppurative myocarditis. The wall of the left ventricle is laid open and the sections separated, showing the presence of an abscess cavity, A. The case was one of puerperal septicæmia.

The parenchymatous variety is also encountered in endocarditis, pericarditis and infections of various forms. The muscular fibres of the parenchyma of the heart undergo a granular degeneration with increase in their nuclei. The muscular structures are softened and present a pale, turbid appearance. These changes are not generally localized. Sometimes the granular change is so extreme that no trace of the striæ can be detected. This is due to the effect of the toxines and is clearly observed in typhoid fever and other infectious diseases, especially when prolonged.

In purulent myocarditis necrosis of the tissues over large and small foci with abscesses are the characteristic features. The abscess formations are in most cases multiple and are liable to rupture into the heart cavities or pericardium. Thus the abscess contents may be discharged into one of the heart chambers, find its way into the circulation and form the starting-points of emboli in other parts of the body. The pressure of the blood upon the wall of the abscess cavity may in turn produce aneurysmal dilatation or rupture. The former contingency is most frequent. The local effect of these abscesses may in rare instances produce profound changes by involving a valve or, by burrowing between the two sides of the heart, result in a fistulous opening. In rare instances the abscess may become encysted and undergo calcification.

Symptoms.—The clinical features of acute myocarditis are those of cardiac enfeeblement. There is lack of any distinctive characteristics. The onset of the affection, as a rule, is insidious and indefinite, although sometimes it is attended with rigors and a pronounced rise of temperature. In some cases there may be nausea, vomiting and pain around the region of the heart. The pulse is small, feeble, rapid and irregular. There is dyspnœa, palpitation, præcordial distress, paroxysms of syncope and sometimes dropsy. When in the course of an acute infectious disease such a combination of symptoms appears, the presence of acute myocarditis may be suspected.

Complications in the form of acute pulmonary affections with bronchitis and pulmonary œdema or pleuritic effusion may occur. Nephritis with diminution in amount of urine excreted and albuminuria may also complicate the condition. Nervous phenomena as in all forms of serious disease of the heart are common.

Among acute affections myocarditis occurs most frequently in diphtheria, where it usually develops somewhat late in the disease, that is, after the sixth day. It is more common among older children and adults. In typhoid fever also the affection is of late appearance; in scarlet fever, it may set in early.

The course of the disease is usually confined to a week. Death commonly occurs from extreme weakness of the heart, but it may sometimes be due to rupture of its walls. In some cases cerebral and pulmonary complications, especially œdema, may be the immediate cause of death.

Diagnosis.—On account of the comparative infrequency of the affection, the similarity of its symptoms to those of other cardiac diseases, and in some instances the marked latency of its clinical phenomena, the diagnosis is difficult and often impossible.

When in the course of a disease with which acute myocarditis may arise as a complication, for example, acute rheumatism, or any of the acute specific or septic fevers, there is rapid development of failing heart power, the presence of acute inflammation of the heart muscle may be presumed.

The differential diagnosis is more theoretical than practical.

Prognosis.—Since the question of diagnosis is vague, that of prognosis must be more or less speculative. In the superficial forms associated with pericarditis and in mild cases complicating typhoid fever the outlook is generally favorable. But in the severe types, especially those arising from septicæmia or pyæmia, the condition is hopeless. These cases usually terminate fatally within a few days. In some death is sudden.

Treatment.—Absolute rest must be rigidly enforced. The patient should lie in the recumbent posture, perfectly quiet, not making the slightest effort.

Remedies directed to combat the adynamic condition constitute the line of treatment, notably arsenic, muriatic acid, iodide of arsenic, rhus, phosphorus and the snake poisons. The diet should be stimulating and nutritious and alcohol should be used freely. For the failing heart strychnine may be administered hypodermically. Digitalis is generally unsatisfactory.

HYPERTROPHY OF THE HEART.

SYNONYMS.—*Simple Hypertrophy; Hypertrophy with Compensative Dilatation.*

Cardiac hypertrophy is an overgrowth of normal tissue in the heart muscle attended with increase of weight. It is an effort of nature to meet and overcome morbid conditions and is therefore a conservative process. There is, however, decided dissent from this view on the part of certain modern physiologists, who regard it essentially as a morbid process. There are two forms. Simple hypertrophy, that in which there is increase in the heart muscle without changes in the chambers, and hypertrophy with dilatation, that in which there is increase in the heart muscle with a proportionate enlargement of the chambers. The latter is also called compensative hypertrophy and corresponds to the old classification of eccentric hypertrophy. The concentric variety described by Bertin is not now recognized. Many modern writers regard it as a post-mortem change.

Hypertrophy may be limited to the walls of a single chamber, to the walls of one side of the heart, or it may involve the entire organ.

When there is obstruction to the general circulation the left ventricle becomes the seat of hypertrophy; when it is in the lungs, either direct or indirect, the right ventricle hypertrophies; when there is stenosis of the auriculo-ventricular orifice the auricles enlarge. Hypertrophy is more pronounced among males than females.

Ætiology.—The causes which give rise to hypertrophy of the heart include those conditions which tend to augment its muscular activity. The active factor is increase of resistance arising from obstructive influences in the circulatory system, the source of which may be in the heart itself, in the arterial circulation, in the state of the blood, in diseases of other organs and morbid conditions of the nervous-system. The increase in size of the heart is approximately in proportion to the additional amount of work put upon it.

The most frequent source of cardiac hypertrophy is the heart itself, namely, the various lesions of the valves and orifices. The abnormal resistance which these conditions exert against the blood-flow would result in more profound disturbance of the circulation, if not speedily minimized by the additional effort of that part of the heart behind the area of resistance. The overworked portion soon becomes hypertrophied and adapts itself, for a time at least, to the altered conditions; hence, compensatory hypertrophy. Thus, insufficiency and obstruction of the mitral valve cause hypertrophy of the right ventricle by increasing the resistance in the pulmonary circuit, and insufficiency and obstruction of the aortic valve cause directly hypertrophy of the left ventricle, which in the case of insufficiency is more marked than in other valvular lesions. In aortic regurgitation sometimes the left ventricle becomes enormously distended, constituting the *cor bovinum.*

With mitral insufficiency additional changes arise. During diastole of the left ventricle there flows into it the accumulated blood of the dilated auricle. This causes the ventricle to dilate and leads to hypertrophy; thus mitral insufficiency causes hypertrophy of both ventricles.

Lesions of the pulmonary valve give rise to anatomical conditions similar to those of the aortic. Both pulmonic insufficiency and stenosis lead to hypertrophy of the right ventricle. Tricuspid defects, which are also uncommon, with the exception of relative insufficiency, cause similar changes, insufficiency leading to right ventricular hypertrophy. It is difficult to define the effect of tricuspid stenosis, as it seldom, if ever, exists alone and its secondary effects have thus not been studied. It would seem, however, that the lesion would result in hypertrophy of the right auricle.

Another source of hypertrophy connected with the heart is the presence of old pericardial adhesions. In the case of adherent pericardium, hypertrophy of both ventricles may result, more particularly the left. The constant accompaniment of valvular defects with adherent pericardium renders it difficult to speak definitely in regard to the influence of the latter upon hypertrophy.

An important group of potential factors includes those conditions which produce increased tension in the vascular system, namely, arterio-sclerosis, dietetic errors, morbid conditions of the blood, disease of the kidneys, pregnancy, muscular strain, diseases of the lungs and deformities of the chest. Arterio-sclerosis, being essentially an affection of advanced years, it follows that the conditions of cardiac hypertrophy to which it gives rise belong to that period of life. In the degenerative processes which characterize its presence the walls of the vessels lose their elasticity and power of osmosis, thus directly increasing the blood-tension. This increase of pressure acts directly upon the heart which undergoes a compensatory hypertrophy similar to that which develops with valvular diseases.

Dietetic errors, by increasing the quantity and altering the quality of the blood, raise the arterial tension and thus lead to hypertrophy. It is claimed that plethora in itself is a sufficient cause and that the ingestion of excessive amounts of food and fluid which augments the volume of blood and even enriches it in hæmoglobin acts in this way. Arguing from the fact that certain lower animals in which nutrition has been crowded for fattening purposes, as, for example, the goose and pig, do not evince evidence of cardiac hypertrophy, it is denied that over-nutrition or plethora alone can act as a cause. There can be no doubt, however, that when to these habits are added those of alcoholic indulgence, they exert an active influence.

The excessive use of alcohol acts in causing arterio-sclerosis by giving rise to abnormally high tension in the circulation and by augmenting the volume of the blood. Of all alcoholic beverages, beer is the most active in the causation of hypertrophy of the heart. This is doubtless due not alone to the effects of the alcohol it contains, but also to the amount of fluid ingested and the presence of carbonic acid gas and the potash salts. Statistics show a greater mortality from diseases of the heart in beer drinkers than in non-beer drinkers. In Germany, the expression "beer heart" is in use, illustrating somewhat forcibly the connection between cause and effect. After beer, brandy, whiskey, wine, coffee and tea, in the order named, act as excitants of hypertrophy, the stronger alcoholics tending to the formation of arterio-sclerosis, the wine to increase in the quantity of the blood. It should be borne in mind that the ingestion of all fluids, even of water, temporarily increases the blood-pressure.

Increased resistance may also arise from morbid conditions of the blood, the result of faulty metabolism, notably in conditions of lithæmia and gout. Diabetes has the same tendency. Nephritis, both acute and chronic, is frequently attended with cardiac hypertrophy. In the acute and subacute varieties it is more common when the lesions are of scarlatinal origin. In these instances there are no chronic arterial changes and the increase in blood-pressure arises apparently from obstruction to the onward flow of blood in the capillaries. The cause of this obstruction is probably the retention of excrementitious matter which acts through the vaso-motor centres upon the middle layer of the vessels.

In chronic nephritis hypertrophy of the left ventricle occurs in both the parenchymatous and interstitial forms of the disease, but is much more frequently encountered in the latter, of which it constitutes an important symptom. In many cases the increase is excessive and includes the musculi papillares. As the condition advances the muscular and fibrous coatings of the arteries become involved and the process of hypertrophy affects the right side of the heart as well as the left. A hard, wiry pulse in the absence of acute febrile disturbances or evident lesion, especially if there is accentuation of the second aortic sound, should always excite the suspicion of interstitial nephritis. The hypertrophic changes of the left ventricle set in early.

Bright attributed the change in the heart wall to either a morbid state of the blood which causes an abnormal stimulus of the organ, or to an increased activity which the heart is called upon to exert in order to propel the blood through distal vessels which have undergone contraction, thus presenting a chemical and mechanical theory.

Since Bright's time, many theories have been advanced to explain the phenomena of cardiac hypertrophy with chronic nephritis. Traube, in 1856, published a paper endorsing Bright's mechanical theory. He regarded hypertrophy as associated with cirrhotic kidney and explained its origin as due to the obliteration of the renal vessels whereby additional work was put upon the heart. Johnson maintained that retention of toxic matter caused the muscular coatings of the arterioles to become thickened and spasmodically contracted, with consequent increase of tension and pressure in the left ventricle. He also held that the walls, not only of the vessels of the kidneys and arterioles, but also of many other tissues were involved in a process of thickening arising from hypertrophy of their muscular fibres.* Hamilton holds that diminished specific gravity of the blood might be the source of obstruction in the peripheral circulation, explaining that the red corpuscles are thereby allowed to mix with the leucocytes, which under normal conditions lie in close contact with the walls of the vessels, and in this way give rise to more or less friction. This theory finds support from analogous conditions in anæmia, especially in the chlorotic form, when the pulse is high and the left ventricle possibly hypertrophied. But in other cases of anæmia there may be low pulse and dilatation of the ventricle. These opposing conditions can be accounted for by regarding the nutrition of the heart muscle in the former to be fairly good and in the latter to be the reverse. In this connection it should, however, be borne in mind that density of the blood is not the only factor which must be taken into consideration in anæmia, for the blood-corpuscles undergo changes which impair their functions to a greater or less extent.

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* Guy's Hospital Reports.

The views of eminent observers on this subject might be quoted at still further length with a corresponding divergence of opinion, but all agree that, whatever may be its causation, the source of hypertrophy of the left ventricle in Bright's disease is some form of increased resistance, the exact nature of which remains a matter of doubt.

Cohnheim maintained that the activity of the renal circulation depends upon the quantity of material for secretion which the blood contains and not upon the absence of sufficient blood in the kidneys, inferring that if any structural change occurred by which the integrity of a part of the kidneys was impaired, a greater amount of pressure would be required, and in consequence greater effort would be called for on the part of the left ventricle. Thus the hypertrophy was, in this sense, compensatory. An important fact which must not be overlooked is that endarteritis obliterans is not infrequently associated with chronic disease of the kidneys.

Pregnancy is a cause of hypertrophy. This fact has been demonstrated by Larcher and corroborated by other observers. The condition is more marked in young and robust women and least so in those poorly nourished. The increase involves both ventricles and the size of the cavities, but is more marked in the left. It begins with the first month of pregnancy and continues up to its termination, when it subsides and a normal condition is re-established. The condition is attributed to the presence of an increased quantity of blood made necessary by the additional demands put upon the circulation for the nutrition of the fœtus, mammæ, uterus and placental circulation.

Muscular exertion is a common cause of hypertrophy and is obviously more frequently observed among those whose occupations require severe and continued physical effort. Among athletes, as is well known, cardiac hypertrophy is also common. Under the influence of unusual muscular exertion the heart may at first respond from its reserve force, but sooner or later the continuous increased efforts result in an augmentation of its muscular structure. In some instances the demands are sudden as well as excessive. This may occur from lifting heavy weights or from some other violent exertion whereby the walls of the heart are strained and insufficiency results. Under these conditions the hypertrophic process which follows is more rapid, and frequently the heart is weakened permanently.

Emphysema, asthma, chronic catarrhal processes, interstitial pneumonia and fibroid phthisis are almost always associated with hypertrophy of the right ventricle. This is pre-eminently true in the case of emphysema. In other instances the dilatation of some of the blood-vessels and the diminution of the calibre of others from compression obviously sets up an obstacle to the blood-current, the inevitable result of which is hypertrophy of the right ventricle. Asthma, chronic bronchitis and interstitial pneumonia are attended with similar results. The conditions known as fibroid phthisis and stone-cutter's lung are notable causes.

Pulmonary tuberculosis seldom causes hypertrophy of the heart, on the contrary, the organ sometimes undergoes atrophy. But in prolonged types of the disease attended with periods of latency with more or less shrinkage and induration of the lung, hypertrophy may develop.

Conditions arising from disturbances of the nervous-system are the source of cardiac hypertrophy. Emotional influences, such as long-continued mental strain and worry, by exciting the heart to greater activity may have this result. Graves' disease is frequently attended with hypertrophy which soon goes on to dilatation. Tachycardia in other conditions or from any cause may likewise lead to dilatation.

Mechanical pressure is an element in the causation of cardiac hypertrophy. This occurs most frequently in deformities attending kyphosis and scoliosis. In pronounced cases of this type there is generally more or less pressure upon the pulmonary artery and its branches which produces obstruction and stasis of the right side of the heart. Pressure from swelling of the bronchial and mediastinal glands may produce the same results.

Congenital contraction of the aorta may prove a source of hypertrophy. Normally the vascular system is in a certain proportion to the size of the individual, but sometimes this is not the case. In these instances the reduction in size of the aorta interposes an obstacle to the flow of the blood.

The respective relations of these ætiological factors to the two forms of hypertrophy may be summarized as follows : Hypertrophy of the left ventricle arises from aortic disease, mitral insufficiency, slow compression of the aorta, chlorosis, atheroma, arterio-sclerosis and Bright's disease. Hypertrophy of the right ventricle, from chronic pulmonary diseases, especially emphysema, mitral and other diseases of the left heart, and obstruction of the pulmonary valve (very rare). Hypertrophy of the heart as a whole from pericardial adhesions, chronic disease of the heart walls, persistent muscular exertion, persistent nervous excitement (Graves' disease), continued action of cardiac poisons, such as alcohol and uric acid, pregnancy temporarily, hypertrophy with compensative dilatation, valvular disease, malformations, adherent pericardium and strain. It is typically observed in aortic and mitral insufficiency affecting the left ventricle and the auricle in incompetence of the auriculo-ventricular valves.*

Morbid Anatomy.—It is not common to find the entire organ involved, the implication of the walls and cavities of more than one chamber being often observed. Sometimes the condition is limited to a very circumscribed area. On account of its more active physiological functions the left ventricle is more frequently affected than the right, the auricles less often than the ventricles and the right auricle more than the left auricle.

The heart undergoes considerable alteration in shape according to the locality affected. In the instance of hypertrophy of the left ventricle the part near the apex is enlarged and the entire organ is elongated, extending to the left and conical in shape. When the right ventricle is implicated the apex loses some of its pointed character, and the organ generally is more spheroid than normal. When the hypertrophy of the right ventricle is excessive the left ventricle is more remote from the chest wall and the apex is represented by the right. The more pronounced the hypertrophy the more decided is the spheroidal appearance. Auricular hypertrophy has little effect on the shape of the organ.

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* Text-Book of Medicine, G. A. Gibson, M.D., Edinburgh and London, 1901.

The structure of the heart is firmer, especially in the case of hypertrophy of the right ventricle, and on section, of darker red, than normal, unless there is some associated degenerative change, in which case it is softer and paler. The musculi papillares and columnæ carneæ are thickened, and are often flattened, particularly in the eccentric form, where there may be increase in the thickness of the septum.

The microscope in many instances shows the change to be due solely to increase of the normal muscular fibres. Whether this arises from augmentation of their number or from simple increase in bulk is an unsettled question. Associated with the hypertrophy of muscular tissue there may be more or less increase of the connective tissue between the muscular bundles, and even between the fibres there may be some newly formed fibrous tissue with numerous nuclei, the result of some interstitial myocardial inflammatory process. Quain designates this condition as false hypertrophy. In this case the color of the heart may assume a grayish hue. Retrogressive changes may be detected in the muscular cells, and the presence in the nuclei of pigmentary deposits may be observed together with granules in the cells. These changes are the result, it is thought, of long continued strain.

Increase of weight in the heart is a reliable evidence of hypertrophy The weight of the normal heart is generally given for males as eleven ounces, and for females nine ounces.* These figures differ from those of Dr. D. J. Hamilton,† who, during a period of thirteen years as pathologist of the Edinburgh and Aberdeen Royal Infirmary, observed the weight, thickness of walls, size of the apertures and length of the ventricles of the hearts of those persons who died in health from traumatism. The number of subjects were twenty-seven males and four females, all over nineteen years of age. The result of these observations gave the average weight of the heart as ten to fifteen ounces, ranging between ten and sixteen ounces; the heaviest weight was observed in the tallest individuals, though there were exceptions. In short persons the heart was generally, but not always, of lighter weight.

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* Quain's Elements of Anatomy, Schäfer and Thane, London, 1892.

† Text-Book of Pathology, D. J. Hamilton, M.D., London, 1887.

The most pronounced degrees of hypertrophy occur in the left ventricles, in some instances being as high as from forty to fifty ounces.

Increase in the thickness of the walls is almost always present. Yet, if the hypertrophy be associated with dilatation, this may be slight. In some cases one part of the walls may be increased in thickness, while the others may be thinner than normal. There may be general dilatation which greatly enlarges the organ without any increase in thickness of the walls, but with enlargement of its chambers. The capacity of the chamber should always be noted, as there may be increase in the size of the wall without appreciable augmentation of its thickness.

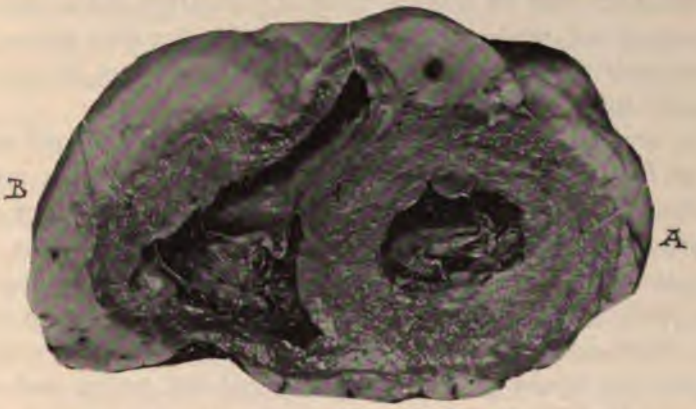

FIG. 32.—Cross-section of heart below the valves of the left ventricle, A, and right ventricle, B, showing hypertrophy and layer of fat.

The dimensions of the cavities and walls in the normal heart are as follows: Left ventricle, 3 to $3\frac{1}{2}$ inches; walls, $\frac{1}{4}$ inch at thinnest and $\frac{1}{2}$ inch at the thickest portion; at the base of the right ventricle, $3\frac{1}{16}$ inches to $3\frac{3}{8}$ inches; wall, $\frac{1}{8}$ inch all over. Diameter of orifices: aortic, $\frac{9}{10}$ to 1 inch; mitral, $1\frac{2}{10}$ to $1\frac{4}{10}$ inch; pulmonary artery, $1\frac{1}{10}$ to $1\frac{2}{10}$ inches; tricuspid, $1\frac{5}{10}$ to $1\frac{8}{10}$ inches.

The ventricular wall may become thickened to the extent of one or even one and a half inches. The walls of the right ventricle may be increased to one inch, the greatest thickness being at the base. The septum does not, as a rule, undergo much change. The walls of the auricles are rarely thickened to more than twice the normal measurement, and if at all involved are usually much dilated.

Symptoms.—The symptoms of cardiac hypertrophy are difficult to define, owing to their frequent association and blending with those of the primary condition and the conservative tendency of the process to correct the disturbances caused by the latter. It is only when hypertrophy is in excess of compensation that symptoms appear. Symptoms pointing to valvular defects may disappear with the establishment of hypertrophy, and so long as this is effective there may be entire absence of circulatory embarrassment during rest and moderate exercise. But this fortunate state of affairs is liable to change, and in time the equilibrium of the circulation may fail to be evenly sustained.

The symptoms which belong to cardiac hypertrophy, and which are at first often intermittent, being excited by exercise, mental emotions, tobacco or alcohol, are a general sense of discomfort in the præcordial region, and a feeling of fullness or constriction. In some instances there may be actual pain. Dyspnœa on exertion is an early symptom, but is not excessive. Palpitation is often easily excited; the heart action is apt to be unduly frequent, accelerated or bounding. The pulse is full and strong and, as a rule, regular, but when degeneration follows its character changes.

The patient may complain of a sensation of fullness and throbbing in the head and neck, flashes before the eyes and ringing in the ears. There may be a dry irritative cough. There is a disposition to hæmorrhages and epistaxis, hæmatemesis and hæmoptysis may occur. Cerebral hæmorrhage may also arise. A large percentage of cases of apoplexy are attended with cardiac hypertrophy.

In hypertrophy of the left ventricle the symptoms of cerebral congestion and the tendency to hæmorrhage predominate. The pulse, in the absence of aortic or mitral lesions, is full and incompressible. High tension of the arterial circulation is commonly associated with left ventricular hypertrophy, and in consequence there is a decided tendency to arterio-sclerosis. At the same time it must be borne in mind that sclerotic changes of the walls of the vessels in many instances precedes and constitutes the origin of the hypertrophic process. In advanced conditions, especially when arterio-sclerosis is the cause of the enlargement, as in Bright's disease, the sclerosed vessels are liable to over-strain and rupture. The most frequent seats of these accident are the brain and lungs.

In hypertrophy of the right ventricle are found the means whereby the tendency to venous stasis and grave phenomena which follow in its course are overcome or mitigated. Dyspnœa on increase of the heart action points to right ventricular hypertrophy and is essentially a symptom of it. When the hypertrophy of the right ventricle is well developed there may be absence of any sign of venous engorgement except in the lungs. In cases with associated mitral and aortic deficiencies the symptoms are those of the primary lesion. The radial pulse does not show any characteristic change. There may be venous pulsations in the neck usually of auricular origin.

When secondary to pulmonary fibrosis or emphysema the condition may be obscured by other disorders. Præcordial distress under these circumstances points to the presence of hypertrophy.

When dilatation develops and compensation fails bronchial catarrh appears and dyspnœa becomes more prominent. The association of arterio-sclerosis may give rise to rupture of the vessels of the lungs, hence hæmoptysis and pulmonary apoplexy may be encountered.

PHYSICAL SIGNS.—These naturally vary according to the part of the heart involved and the degree of enlargement.

Left Ventricle.—Inspection, in females and in children, shows some bulging of the præcordial region with broadening of the interspaces. The apex-beat covers a wider area than normal with displacement downward and to the left. The force of the impulse appears very strong.

Palpation verifies the signs of inspection and shows that the apex may be displaced as far down as the seventh interspace and as far to the left as the anterior axillary line. The impulse is strong and forcible and is generally described as heaving. In uncomplicated hypertrophy it is strong and regular and of the normal rate of beat. When much dilatation is present it is more abrupt and less forcible.

Percussion shows the area of dulness to be extended, both laterally and vertically. It may extend upward as far as the second interspace and to the left one or two inches beyond the mid-clavicular line. When there is associated hypertrophy of the right ventricle it extends also to the right.

Auscultation discloses various changes. In simple cases there is usually prolongation and unusual fulness of the first sound with accentuation of the second. The degree of intensity varies according to the condition of the ventricle and blood-vessels. There may be a peculiar metallic clink to the right of the apex-beat. The second sound may be doubled. This is due to high arterial tension. When there is decided dilatation, the second sound becomes more feeble, while the first, on the other hand, is intensified. The presence of valvular lesions necessarily greatly modifies these sounds.

Right Ventricle.—In young subjects and females inspection may show bulging in the region of the sixth and seventh ribs and of the lower portion of the sternum. The apex-beat is diffused and may be observed in the sixth interspace close to the border of the sternum. In thin persons there may also be pulsation between the ensiform cartilage and seventh rib, to the right of the sternum and in the third and fourth interspaces, especially in eccentric hypertrophy.

Palpation does not add to these signs except to confirm them.

Percussion shows enlargement of the normal area of dulness an inch or more beyond the right border of the sternum. Excessive enlargement transversely points to the predominance of dilatation over hypertrophy.

Auscultation in simple hypertrophy shows slight prolongation of the first sound and accentuation of the pulmonary second sound. Owing to the condition of vascular tension in the pulmonary circulation, the second sound of the pulmonary valves may be doubled.

The presence of emphysema greatly modifies the heart-sounds and may even render them negative.

Hypertrophy of the left auricle does not give direct evidence of its presence by physical signs. It may be assumed to occur with mitral obstruction and insufficiency. Relative dulness in the second left intercostal space and a distinct impulse preceding the apex-beat are mentioned by some observers.

Enlargement of the right auricle is more common than that of the left and much more readily observed. It is more of a dilatation than a hypertrophy. Systolic jugular pulsation, dulness to the right of the sternum in the third and fourth interspaces, with sometimes a wavy impulse preceding that of the apex, are the signs of its presence. The condition is secondary to tricuspid insufficiency and enlargement of the right ventricle.

X-RAY DIAGNOSIS, see Part VII.

Diagnosis.—The recognition of hypertrophy of the heart may be summarized as depending upon the physical signs with the corroborative evidence afforded by the ætiology and objective symptoms. Hypertrophy of the left ventricle may be known by the increased force of the heart, its heaving impulse, together with the increase in depth of tone and prolongation of the first sound with accentuation of the second.

In hypertrophy of the right ventricle the increased area of dulness to the right and change in position of the impulse are points of recognition. When extensive emphysema is present it is often difficult to establish the diagnosis except by inference.

The frequent association of dilatation with hypertrophy renders the question as to which predominates of importance. This can only be determined by carefully comparing the results of physical examination and the objective symptoms. In hypertrophy there is increased vigor of the heart's action, while in dilatation it is slower and more feeble and frequently irregular. When dilatation first appears in the left ventricle there is change in the position of the apex-beat on inspection and palpation. Again, the strong, heaving impulse of hypertrophy is distinguished from the sudden shock of commencing dilatation and the consequent increased rate and irregularity of the pulse.

From aneurysm of the arch of the aorta cardiac hypertrophy is to be recognized by the fact that the enlargement in the latter is always upward either to the right or left; from left-sided sacculated pleuritic effusion, by the absence of expansion during inspiration, by the history of the case, by displacement of the apex-beat upward and later by its absence, by muffling of the sounds of the heart and often the presence of a friction rub; from tumors of the mediastinum, by the fact that the area of dulness is upward and the displacement

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to give rise to its presence. Exceptions to this are not infrequent, as in the case of general arterio-sclerosis.

Cases arising from acute or subacute nephritis are frequently restored when the kidneys recover, while those occurring in connection with pregnancy disappear with the termination of that condition. It is, however, in its relation to chronic processes that cardiac hypertrophy demands the most important consideration.

When hypertrophy arises from valvular disease the prognosis, while largely influenced by the concomitant conditions discussed, is essentially that of the individual lesion which it accompanies. When associated with arterio-sclerosis the prognosis must be guarded on account of the possibility of myocardial degeneration and the liability of rupture of the walls of the blood-vessels which may be looked for in connection with this process. In Bright's disease the conditions are similar, but are more progressive. In arterio-sclerosis, as age advances, the failing power of the heart may exert a conservative process by lowering the pressure within the blood-vessels and thus lessen the chances of their rupture.

The indications as to prognosis may be summarized as follows: If in a case of cardiac hypertrophy the patient suffers from dyspnœa, with rapid and irregular and sometimes feeble pulse, and a tendency to œdema, a group of symptoms are present which must be regarded as a positive indication that dilatation predominates over hypertrophy, and that the outlook is unfavorable.

Treatment.—When hypertrophy is adequately compensative the chief aim is to keep it so. This can only be accomplished by the observance of care in diet and mode of living. Excesses of all kinds should be avoided, and the general directions as considered under the subject of valvular diseases when the lesion is fully compensated should be enjoined.

When the organ shows evidence of over-activity or over-hypertrophy, as it is sometimes called, and cerebral and thoracic symptoms manifest themselves, greater care will be demanded and all influences which tend to stimulate the irritable and over-excited heart should be guarded against. The diet should be nutritious, but the more concentrated foods should be used sparingly, and the daily quantity taken should be rather less than that in health.

Anything, on the other hand, that tends to depletion or lowering of the system should be interdicted. Anæmia, prolonged lactation, severe purgation and diarrhœa are conditions which must be carefully avoided, likewise indigestion and flatulent distention.

The amount of fluid should also be decreased and the diet made as dry as compatible with health in order to reduce the tendencies to intra-cardiac pressure. Alcoholics, tea, coffee and tobacco should be forbidden. Exercise should be of a mild nature, and any that tends to excite the heart should be forbidden. Some patients are benefited by gentle exercise, while in others it has a contrary effect. When the extremities are cold Hale recommends exercises which only affect the legs and arms, while the trunk remains quiet. The bowels should be kept open not only to obviate the disturbing influence of intestinal torpor, but also to prevent straining at stool.

While remedies can play no part in the treatment of hypertrophy *per se*, many of the symptoms are amenable to relief. The following are suggested :

Aconitum is recommended for the congestive symptoms. It has been condemned for its depressing effect on the heart, but this was doubtless due on account of too free dosage. The remedy should be administered in fractional doses of the tincture or in dilution.

Nitroglycerin may be given when the arterial tension is high, especially in arterio-sclerosis, for the sense of præcordial weight and discomfort, tinnitus aurium, sense of fullness in the head and vertigo.

Belladonna has indications similar to nitroglycerin, instead of which it may be used when there is cerebral congestion with flushed face and throbbing in the heart. It is indicated in strong, full-blooded persons.

Cactus grandiflorus is useful when the characteristic indication of a sense of constriction is present. It is also useful for palpitation, worse at night and when lying on the right side.

Lilium tigrinum is especially recommended by Goodno. Its cardiac indications are, dull, oppressive pain in the præcordial region, worse at night, sensation of constriction around the heart, alternating with relaxation, sense of oppression, fluttering and palpitation.

Rhus is said to be useful in conditions of hypertrophy from over-exertion without valvular defect.

Veratrum viride has the same general indications as aconite, but is to be preferred when the heart power is stronger and the symptoms of thoracic and cerebral congestion are intensified.

Gelsemium is useful in conditions of passive cerebral congestion.

Aurum in the form of the chloride of soda or the metal in trituration is indicated in cases attended with arterio-sclerosis with strong action of the heart and tendencies to visceral congestion.

Digitalis may be used in the first, second or third centesimal dilution, never stronger.

Chamomilla, coffea, caffeina, ignatia, the bromides and valerian find their sphere of action when nervous symptoms appear.

Other remedies which are suggested for consideration are plumbum, spigelia, kalmia, prunus Virginiana, bromium and arsenicum.

The selected remedy should be administered about four times a day and continued for some time. When it appears that dilatation is predominating and signs of weakness develop, the therapeutic indications are radically different, being those of dilatation.

DILATATION OF THE HEART.

Dilatation of the heart signifies enlargement of one or more of its cavities without a proportional increase in the strength of its walls. Two varieties are recognized: that in which the enlargement of the cavities is accompanied by an increase in the thickness of the walls and that in which it is accompanied by a thinning of the walls. The former is much more frequent. A third variety is described by some writers, namely, dilatation without appreciable mural changes, but this is apparently more a matter of theory than of fact.

Dilatation with increased thickness of the heart walls, otherwise, dilatation with hypertrophy, is not identical with hypertrophy with dilatation, although the latter frequently merges into the former.

In dilatation with hypertrophy the cavities frequently become greatly enlarged, while the walls, although attaining an abnormal thickness, on account of the presence of degenerative processes, do not acquire a corresponding strength, and in consequence are unable to meet the demands made upon them. In hypertrophy with dilatation, similar enlargement of the cavities ensues, but the mural enlargement is equal to the additional stress put upon it. In all instances of hypertrophy, however, no matter how completely compensated the process may appear, there is always a possibility of its breaking down and of dilatation predominating.

In dilatation with thinning of the walls, the change may be slight, the cavity being only moderately increased in size, with a corresponding change in the walls. Cases of this nature may be seen in prolonged fevers. On the other hand, the process may be excessive, the wall being almost unable to contain the blood. Dilatation is nearly always attended with some hypertrophy, hence the latter term is employed in a relative sense. Dilatation with thinning of the heart walls may arise directly from the effects of intra-cardiac pressure or it may be superimposed upon a compensatory hypertrophy; that is, the latter breaks down and the element of dilatation prevails.

Dilatation may manifest itself either with acute or with slowly appearing chronic symptoms, according to the cause.

Ætiology.—The influence of heredity is frequently observable, as evidenced by the predisposition among certain families to degeneration of the heart walls.

This tendency is more likely to be operative in persons of a gouty or rheumatic diathesis. As in hypertrophy, dilatation is more frequent among males than females, on account of the former being more exposed to the causal influences. It is also more frequent during adolescence and old age. This is doubtless due to the fact that the changes which the organism undergoes at puberty afford a freer play to any disposition to cardiac disease, while in old age the proneness to degenerative processes is an accompaniment to that period of life.

The cardinal factors in the ætiology of dilatation are intra-cardiac blood-pressure and loss of strength of the walls; either may be the sole cause, but more frequently both act conjointly. Behind both these conditions stands an array of morbid processes and influences. Those which may be regarded as arising from intra-cardiac pressure include lesions of the valves and orifices. The result of valvular and orificial defects in all instances is dilatation, which, if the walls are healthy, is immediately attended with a process of compensative hypertrophy and for a time, at least, all evidences of cardiac disturbance and circulatory embarrassment are everted or removed. But if, on the other hand, the walls have not sufficient power to meet the additional strain put upon them, dilatation with thinning ensues, followed in turn by symptoms of failing heart power. Illustration is furnished in the instance of aortic stenosis. Here the obstruction at once gives rise to dilatation of the left ventricle from the increased blood-pressure. This is quickly followed by hypertrophy with compensation. In time, however, the heart muscle becomes impaired, compensation ceases to be effective and secondary dilatation follows. The relations of valvular disease to dilatation have been considered in detail in the discussion of the various individual lesions.

The other factors which give rise to dilatation from intra-cardiac pressure are arterio-sclerosis, muscular strain, dietetic errors, disease of the kidneys, deformities of the chest, neuroses and certain pulmonary affections, such as fibroid induration of the lungs, emphysema and asthma. The same conditions, it will be observed, which enter into the ætiology of hypertrophy.

Among special causes acting upon pre-existing tendencies, Broadbent mentions injudicious hydropathic treatment, the Banting method for reducing obesity and inhalation of Himrod's asthma powder. Adherent pericardium may also give rise to dilatation, though it is not a frequent cause.

Acute dilatation from intra-cardiac pressure is not uncommon. Muscular efforts of any kind, climbing, lifting or hard work, long marches, strong emotional influences or an alcoholic debauch may, by increasing the intra-ventricular tension, suddenly give rise to dilatation, or it may cause a condition of dilatation with compensative hypertrophy to become acutely inadequate. Again, compensatory hypertrophy may become impaired by recurrent attacks of endocarditis.

In acute or subacute nephritis, dilatation often occurs both with and without hypertrophy. In chronic renal disease it forms an essential symptom, especially in interstitial nephritis.

The second great factor in the production of dilatation, namely, weakness of the wall, may result from intra-cardiac pressure or may occur independently of it. The resistive force of the walls must be taken in a relative sense. They may be proportionately too weak to resist a normal or even a subnormal pressure, as well as be inadequate to the force of the augmented intra-cardiac tension. For example, in low fevers and depleted conditions of the system the blood-pressure may be low, yet the heart wall may become too weak even to afford effective resistance to the lowered pressure.

The causes which impair the integrity of the walls are both acute and chronic. The former includes acute peri- and endocarditis, especially when these inflammations are rheumatic, and acute specific fevers, notably scarlet fever, typhoid and diphtheria, and occasionally muscular strain from over-exertion. In infections the active factor is probably the presence of toxines in the blood.

By far the most frequent form of mural deterioration is that in which the processes are chronic. Here are encountered numerous causes, many of which have been mentioned as operative in connection with the production of increased tension. These influences may be summarized as follows: Malnutrition of the myocardium from any cause, especially disease of the coronary arteries, chronic endocarditis, faulty assimilation and metabolism, defective nutrition from poor living or over-feeding, alcohol, drugs, uric acid the effects of tobacco, continuous overstrain, prolonged nervous influences which depress the cardiac ganglia, anæmia and long continued exhausting illnesses. In short, all conditions which tend to give rise to fatty and fibroid degeneration, arterio-sclerosis and muscular weakness.

There are some cases to which it is difficult to assign any cause, and which in consequence are called idiopathic. The symptoms appear suddenly and acutely. There is dyspnœa, cough and great cardiac distress. Some terminate fatally in a few days, while others run a chronic course.

Morbid Anatomy.—The enlargement of the organ, the relative proportion of the thickness of the walls to the size of the cavity, the weight and the tissue changes which the myocardium undergoes are the salient points in the morbid anatomy of dilatation.

Either one cavity of the heart may be dilated or the whole organ may become involved; the latter is the most frequent. The part upon which the causal influences most actively exert their force generally manifests the greatest amount of change.

The gross appearance shows changes in shape and increase in bulk varying according to the part involved. It is more spherical when all the cavities are involved and broadened at the apex when the ventricles alone are dilated. When the left ventricle is the chief seat of the process it is longer and larger; when the right is mainly affected the transverse diameter is increased, the organ is more globular anteriorly than it should be and the apex is more obtuse. In some instances the enlargement may entirely hide the normal apex and form in itself the apex. When the auricles are dilated they constitute large masses on each side of the aorta and pulmonary artery.

Where dilatation arises secondary to valvular disease generally more than one chamber of the heart is affected. The relative frequency of involvement of the several chambers is as follows: The right ventricle rather more than the left, the auricles, especially the left, more than the ventricles, hence it follows that the left ventricle is least susceptible to dilatation, although it undergoes more hypertrophy.

In aortic insufficiency there is almost always marked dilatation of the left ventricle with a corresponding degree of hypertrophy, but as the coronary arteries are frequently unable to meet the increased demands of nutrition, dilatation preponderates. In obstruction of the aortic orifice there is less tendency to dilatation with thinning of the walls. the principle change being dilatation with compensative hypertrophy of the left ventricle. This condition continues for a time, when degeneration and dilatation result together with implication of the mitral valve.

In mitral insufficiency the left ventricle is dilated to a high degree, but there is a corresponding hypertrophy which tends to prevent undue dilatation for a long period; under these conditions the left auricle is the seat of extensive dilatation. In mitral obstruction there is hypertrophy of the left auricle which may give way to extreme dilatation. The left ventricle generally becomes more or less hypertrophied followed by hypertrophy of the right. The right ventricle is generally involved as the result of lesions in the left side of the heart. Aortic disease has a much less pronounced effect on the right side of the heart unless the left ventricle is enfeebled, when the same conditions obtain as in the case of mitral disease. Mitral lesions, especially insufficiency, affect the right ventricle very powerfully by impeding the pulmonary circulation. In these conditions there is generally dilatation of the right side of the heart.

In pulmonary affections which are prone to cause dilatation the right ventricle is sometimes dilated and hypertrophied to an excessive degree.

In pulmonic valvular deficiencies the right ventricle becomes both dilated and hypertrophied; in the case of obstruction the latter is more prominent; in insufficiency, the former. In tricuspid lesions the right ventricle dilated in insufficiencies, while in obstruction it chiefly undergoes hypertrophy.

As when the general circulation is obstructed the left side of the heart suffers, so when the pulmonary circulation is obstructed the right side of the heart becomes involved. In cases of interstitial nephritis, arterio-sclerosis and in all instances where, from any cause, the peripheral circulation offers resistance to the free discharge of blood from the left ventricle, that chamber dilates and hypertrophies. The effects of strain and emotions are similar to obstruction.

When dilatation is excessive the heart walls may stretch the auriculo-ventricular rings and relative insufficiency ensue.

The weight of the heart during dilatation is generally increased. In so-called simple dilatation there is said to be no change. But, as stated previously, this variety of the lesion is scarcely a practical entity, for there is almost always some hypertrophy with every form of dilatation.

The size of the heart chambers and thickness of the walls have been given at length in the article on hypertrophy to which the reader is referred. It is often difficult, however, in post-mortem examinations, to determine the relation between the size of the cavities and the thickness of the walls. This is owing, in part, to changes after death and to the condition of the heart at death, whether it was in systole or diastole. (Bristowe.)

The state of the muscle structures vary according to the amount of dilatation and the extent and character of any degenerations which may be present. Unless the latter is very decided and the dilatation excessive, the organ is flaccid and either normal in color or somewhat pale. Sometimes it is mottled. When, however, dilatation is extensive and degeneration correspondingly so, it presents the appearance, both gross and microscopic, discussed under fatty degeneration and fibroid degeneration according to which the condition prevails.

The consequences of dilatation are as profound as they are widespread and comprehensive. Its presence is a confession on the part of the heart that it is no longer able to effectively perform its great function of propulsion of the blood. Its chambers fail to empty themselves completely and, at the same time, fail to permit a normal amount of blood to enter. Thus the arterial system no longer receives its normal supply, while the venous becomes congested and stagnated. It is not difficult to see how, under these conditions, pathological changes in distant organs are induced and how the already defective nutrition of the heart is further aggravated. The coronary arteries become inadequate to sustain the heart, either from impoverishment by the process of dilatation or from lack of power to meet the increased demands. As a result degenerations follow. Again, on account of the backward pressure on the coronary sinus, there may be an excessive development of fibrous tissue attended with interstitial myocarditis.

When the process originates in the left ventricle dilatation and distention of the left auricle soon follow. The blood in the pulmonary veins thus finds itself impeded and as a result stasis of the pulmonary circulation with the attendant phenomena of bronchial catarrh, dyspnœa and hæmoptysis make their appearance, at the same time the pulmonary vessels not infrequently become sclerosed. The obstruction of the pulmonary flow next reacts through the pulmonary artery upon the right side of the heart, the ventricle and auricle of which respectively undergo dilatation. Then there follows, with the inability of the right ventricle to empty itself, stagnation of the entire venous circulation. The obstructive influence of backward pressure next exerts itself upon the great veins and the jugulars at the neck become greatly distended. As a consequence of its close connection with the inferior vena cava the hepatic circulation becomes involved. The pressure in the hepatic vein and radicles is attended with distention and engorgement of the liver, with increase of fibrous tissue between the lobules and around the vessels, and fatty degeneration of the cells. This is the condition commonly known as nutmeg liver. In some cases the development of the fibrous tissue is followed by atrophy of the organ. The next retrograde step is obstruction of the whole portal system as a consequence of the damming back of the capillary flow in the liver. The stomach, spleen, intestines and peritoneum now share in the process of stasis. Gastric and intestinal catarrh with enlargement and fibrous induration of the spleen are added to the morbid manifestations. The peritoneum passes into a condition of passive hyperæmia and transudes a large amount of serum. The kidneys likewise fall into a state of disorganization, becoming congested, enlarged, more friable and the capsule more easily detachable than normal. As the process advances they become indurated with more or less shrinkage and adhesion of the capsule. The brain may likewise feel the effects of the altered circulation. Its cortical substance may undergo some diminution, while its veins may show evidence of engorgement. Dropsy appears comparatively early around the ankles and gradually extends upward. The subcutaneous tissues of the abdomen become infiltrated and the peritoneal cavity more or less filled with fluid.

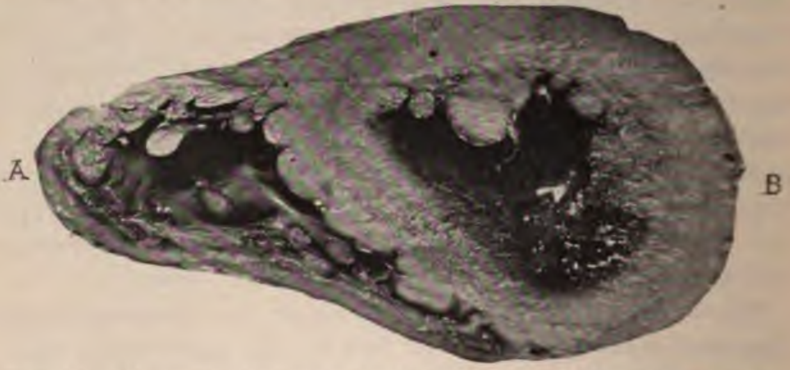

FIG. 33.—Cross-section of heart showing dilatation of the right ventricle, A, and dilatation and hypertrophy of the left ventricle, B.

Symptoms.—In acute dilatation symptoms appear suddenly. The patient suffers from palpitation, dyspnœa and præcordial distress, which sometimes amounts to pain. The pulse is rapid and compressible. In severe forms there is distention of the veins of the neck and temples, with more urgent dyspnœa and œdema of the subcutaneous tissues of the extremities and abdomen. The pulse, in such cases, may be as high as one hundred and sixty or one hundred and eighty per minute. When acute dilatation occurs in connection with acute infectious fevers, the symptoms usually speedily become grave.

Chronic forms of dilatation manifest themselves in most instances very insidiously. In the early stages there is breathlessness on slight exertion and more or less palpitation. Accompanying these symptoms there is a sense of oppression and weight in the region of the heart, especially if dilatation is pronounced. As the lesion advances dyspnœa on exertion becomes excessive. The patient is not only unable to go up-stairs, but can scarcely walk on a level without oppression. Even in repose respiration is more or less labored. At night the dyspnœa frequently becomes aggravated, and sleep and the recumbent position are impossible. Occasionally, however, although extremely ill, some persons are able to lie down. Apart from the disturbance of dyspnœa, insomnia is frequently very troublesome. The countenance wears an expression of suffering and distress, the lips are bluish, the eyes watery with puffiness underneath, and the capillaries of the cheeks are injected. When obstruction to the circulation is excessive the face is cyanosed and the great veins of the neck and temples stand out in a state of engorgement, and the slightest movement aggravates the difficulty in breathing. The extremities are cold and pale or purple and livid, especially if pendant. The pulse is frequent, often irregular, intermittent and generally of feeble tension. The urine is high colored, scanty and loaded with urates, and contains albumin and casts. The hyperæmia of the lungs tends to bronchitis, bronchial catarrh and cough, which is often annoying. Gastro-enteric symptoms are common, there is loss of appetite, indigestion, constipation and diarrhœa. The liver becomes engorged and enlarged, sometimes painful and pulsating. In some cases there is a decided icteric hue from catarrh of the bile duct and pressure upon it. Cerebral symptoms may appear in the form of somnolence, apathy and wandering, especially on waking. The patient also complains of distressing dreams.

Dropsy is a very important and significant symptom. In the earlier periods of dilatation it may confine itself to the ankles for some time, but later it creeps up and involves the thighs, genitals and abdomen, at the same time the hand and forearm become affected and ascites appears.

PHYSICAL SIGNS.—*Inspection* in the case of dilatation of the left ventricle reveals a feeble, fluttering, undulatory impulse. The apex-beat shows decided weakness or is not visible. When the right ventricle is much dilated its apex impulse replaces that of the normal impulse. This is due to the enlargement which may overshadow the left ventricle and which brings the right ventricle in closer contact with the chest wall. The impulse then is seen below the ensiform cartilage or, less commonly, to the right or left of it. A wavy motion is observed in the fourth, fifth and sixth interspaces to the left and close to the sternum.

In extensive dilatation of the right auricle there is pulsation in the third right interspace. Pulsation in the second left interspace is probably due to throbbing of the conus arteriosus, and if there is tricuspid regurgitation a systolic murmur may be heard.

A prominent feature in dilatation of the right ventricle is distention and pulsation of the jugulars. They may, however, be distended without pulsation. In very thin persons the whole præcordial space may seem to have a wavy motion. In stout persons inspection may be entirely negative.

Palpation confirms the feebleness of the heart action and diffuseness of the impulse, but usually fails to reveal the apex-beat. Walshe pointed out that when there is an apex-beat it may sometimes be seen, but cannot be felt. A wide area of impulse may be present with a weak apex-beat. Sometimes a thrill may be appreciable.

Percussion shows general increase of the area of dulness, especially to the right and left of the normal area. In the case of dilatation of the left ventricle, dulness is increased to the left, to or even beyond the mid-clavicular line, up as far as the second rib, downward sometimes as far as the sixth and in very rare cases beyond it. In dilatation of the right ventricle there is increased dulness to the right to the extent of about one inch or more beyond the right of the sternum on a line with the fourth interspace.

When both ventricles are involved the area of dulness partakes of the above directions, showing bilateral increase in a somewhat transverse direction. The shape of the area of dulness is oval or relatively square. The presence of emphysema may greatly modify the signs of percussion.

In dilatation of the right auricle there is dulness on percussion to the right of the sternum in the third and fourth interspace.

Auscultation finds the sounds of the heart weakened in intensity and changed in character. The first sound is shorter in duration and higher in pitch. In pronounced cases it is weak and very like the second sound. The second sound is frequently inaudible at the apex, and the period between the two sounds is shorter on account of the accelerated heart action. In some instances the second sound may be louder than the first. It is, therefore, sometimes difficult to distinguish one from the other, especially when the pause between them is shortened. The sounds of the heart under these conditions have been compared to those of the fœtus.

In dilatation of the left ventricle alone the pulmonic second sound may remain strong provided the right ventricle has not undergone much dilatation and compensation is unbroken. When the left ventricle is much dilated the aortic second sound becomes weak.

Reduplication of sounds may occur, but is uncommon. Some observers speak of reduplication of the first or second sound which gives rise to the galloping rhythm, others of an apparent reduplication of the first sound and an actual doubling of the second.

Auscultation also discloses interesting and important phenomena in regard to murmurs. In the greater number of cases an auriculo-ventricular systolic murmur is present. When dilatation is excessive or is rapidly becoming so, a pre-existing murmur may disappear. On the other hand, dilatation may develop murmurs by stretching the auriculo-ventricular orifices and causing relative insufficiency. Again, when dilatation has improved under treatment, a previous murmur which had disappeared may reappear with clearness.

X-Ray Diagnosis, see Part VII.

Diagnosis.—A weak, irregular heart, an extended undulatory impulse, small, feeble, irregular or intermittent pulse, weakness or entire loss of the apex-beat, increased area of dulness laterally, resemblance of the first sound to the second, decrease of the interval between the first and second sound, symptoms of pulmonic and general congestion are the features which characterize dilatation. The history will add much to the situation.

The condition may be mistaken for hypertrophy, pericarditis with effusion, consolidation of the lung near the heart, aneurysm and thoracic tumors.

In both dilatation and hyperthrophy there is a diffuse wavy impulse on inspection, and increase in the area of dulness, but dilatation is characterized by feebleness in its heart-sounds, by frequent absence of the apex-impulse, the resemblance of the first sound to the second and by a rapid, weak, intermittent or irregular pulse, a direct contrast to the vigor of hypertrophy with its slow, heaving heart motion, its full, regular pulse, abnormal position of the apex-beat and prolonged dull first and accentuated second sounds. In addition to the physical signs, the history of the case and the rational symptoms will point to the diagnosis. For the differentiation between dilatation of the heart and pericarditis with effusion see page 46.

Consolidation of the lung in close proximity to the heart may give rise to some of the signs of dilatation, but may be differentiated by the absence of the characteristic changes in the heart-sounds which belong to dilatation, and by the attending signs and features which accompany pulmonary changes.

The enlargement of dilatation and hypertrophy may be distinguished from that of aneurysm and of tumors in the thorax by the fact that the increased area of percussion dulness is referred to the right, while in enlargement of the heart it is bilateral and generally downward. As far as aneurysm is concerned, the other physical signs of its presence are generally sufficiently characteristic to prevent error.

Prognosis.—In dilatation arising in connection with the acute infectious fevers the outlook is very grave. Acute dilatation superimposed upon hypertrophy with dilatation, if caused by some extraneous influence, such as over-exertion, while always serious, in many instances is amenable to treatment. In those forms occurring as a result of a giving way of compensation with degeneration of the walls, the forecast is decidedly unfavorable. Frequently, however, much may be done in the way of care and treatment to sustain the failing heart power and to prolong life. In conditions attended with atheroma and those in which there is reason to suppose that fatty degeneration is present, the prognosis is bad and the possibility of sudden death must be borne in mind. An irregular pulse, the disappearance of previously existing high tension without treatment, feeble, fluttering impulse, are unfavorable indications. Paroxysms of dyspnœa and attacks of syncope are dangerous. Many perish in this way, but the most frequent cause of death is pulmonary œdema.

Treatment.—The application of remedial measures to cardiac dilatation must be viewed from the standpoints of removal of the cause and management of effects. In all cases efforts should be made to ascertain the ætiological influences; in a large number of instances, however, these are irremediable and beyond the sphere of therapeutic agencies, as, for example, valvular diseases. On the other hand, there are conditions in which the causes are removable when the surroundings and means of the patient permit. These are mental and physical stress, anæmia and excessive use of alcoholic beverages. Those who are subjected to continuous mental strain and worry should be removed to different surroundings and scenes. Those who suffer from the effects of physical exertion should seek rest or occupations which do not call for muscular efforts. Those whose constitutions are debilitated and whose blood is impoverished should be toned up by highly nutritious food, iron, arsenic and remedies of that class. The use of alcoholics, except under medical supervision, should be forbidden, likewise tobacco, coffee and tea.

When pulmonary disease is the starting-point of dilatation, every means should be employed to control its progress. Efforts to relieve the bronchial catarrh and emphysema and the extension of pulmonary lesions should be instituted. Dilatation depending upon valvular diseases should be treated according to the principles laid down in the consideration of that subject.

Standing at the head of the list of remedial agents are rest and nutrition, the importance of which cannot be too forcibly emphasized. Prolonged rest in bed should be enjoined, the length of time depending upon the exigencies of the case. In not a few instances gratifying results follow. A very rapid pulse may become reduced in frequency and improved in strength, the area of heart dulness diminished, a pre-existing murmur may return and dyspnœa and other symptoms of congestion relieved. Especially may this result be observed in those cases where a condition of dilatation comes on somewhat acutely from the effect of over-exertion or similar causes in persons suffering from valvular deficiencies. After removal from bed the patient should be made to recline on a lounge or reclining-chair and after a suitable interval may be allowed to go about, but only on a level. Stairs, sudden exertion and mental annoyance must be avoided. The bowels should be kept free, if for no other reason than to prevent straining at stool.

The diet should be nutritious, concentrated and as dry as possible. Food should be taken in small quantities at regular intervals four or five times a day rather than heartier meals less frequently. Nitrogenous food should be given freely. In the case of patients of gouty or rheumatic diathesis the diet should be modified accordingly.

In conjunction with rest, massage often proves a valuable adjuvant. It should be general, but not administered sufficiently long to fatigue the patient. In those persons in whom it produces a condition of nervous irritability or exhaustion it should be discontinued.

Of equal importance is the administration of drugs, among which digitalis is chief. There is no other remedy which will be so frequently called for in dilatation. Other remedies of the digitalis group are also of value, notably strophanthus. Strychnia, the iodides of arsenic and of potash and nitroglycerin are also of service. The special indications for the administration of these remedies are discussed in the sections on general cardiac therapeutics, and in that on the treatment of failing compensation in valvular disease.

In suitable cases Schott's method of resistance exercises and Oertel's system of walking up graded inclines, together with the baths of Nauheim, may produce satisfactory results. Conditions of dilatation due to strain or over-exertion, atheroma or renal disease are not to be subjected to these methods, but dilatation arising as a consequence of a general atonic condition, with or without valvular defect, cases of early date and those associated with obesity are likely to undergo decided improvement.

When dilatation has advanced to such a degree that general systemic disturbance begins to be manifest and the long train of distressing phenomena which betoken a failing heart appear, treatment must obviously be on the line of palliation for the relief of suffering. The symptoms under these conditions are essentially those of the latter stages of valvular disease and the treatment is identical.

ATROPHY OF THE HEART.

Atrophic changes involving the heart may be either local or general. In the former only one chamber or a part of the heart wall at any point or the papillary muscles may be affected. The walls of one ventricle, for example, may become greatly reduced from abscess or aneurysm. The most frequent source of local atrophy is mitral obstruction which gives rise to wasting of the left ventricle, and myocardial degeneration in association with fibrous overgrowth.

Congenital atrophy, or, more properly, arrest of development of the heart also occurs. When observed it is found more frequently among females with non-development of the sexual organs and arterial system with general faulty nutrition. This condition is sometimes attended with pronounced types of chlorosis, as described by Virchow.* An abnormally small heart, however, may be compatible with health, as occasionally on post-mortem examinations on persons dying of acute disease a heart out of all proportion to the size of the body has been found.

It is the purpose of this article to consider general atrophy and emaciation of the heart and not any of the several local atrophic processes which more properly are part of the affections to which they are secondary and of which they form a more or less essential accompaniment.

Corresponding to the old classification of hypertrophy, earlier writers spoke of simple, eccentric and concentric atrophy; simple, implying reduction in weight from thinness of the walls; eccentric, thinness of the walls with increase of the size of the chambers; and concentric, that in which there is both reduction in the size of the organ and of the walls. These divisions are not altogether practical as there is doubt as to their actual existence. Eccentric atrophy properly belongs to the subject of dilatation and is simply a condition of a dilated heart. Pepper says that weight alone decides the question of the presence or absence of atrophy whatever the size of the organ.

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* Ueber die Chlorose, Berlin, 1872.

Ætiology.—The common cause of general cardiac atrophy is some wasting disease which follows a protracted course, such as tuberculosis, diabetes, cancer, syphilis, Addison's disease, etc. The condition also occurs in old age, in association with amyloid degeneration of the kidneys and in the insane. Local sources of origin have been observed in adhesive pericarditis and calcification of the pericardium. Disease of the coronary arteries is generally mentioned as a cause, but this condition is much more likely to result in degenerative changes, rather than simple atrophy. A rare cause is excessive fatty deposit which may give rise to wasting of the heart muscle.

Morbid Anatomy.—The main feature in the pathological process is reduction in weight. The normal heart in the adult is roughly estimated as being about the size of the fist, weighing in the male from ten to twelve ounces and in the female from eight to ten. Sometimes the reduction from the normal is excessive. Quain relates a case of a girl of fourteen in whom the heart was found to weigh one ounce and fourteen drachms, and Bramwell, one in which it weighed two ounces and two drachms.

In addition to diminution in size the heart shows absence of the superficial fat, rendering its contour less rounded and the vessels more pronounced. When the shrinkage is excessive the visceral layer of the pericardium may be thrown into folds on the surface. The appearance of the heart also shows decided variations according to the presence or absence of fatty degeneration and pigmentation. The muscular substance may appear either normal, or paler or darker. In consistency it may be softer or more dense. In cases of simple atrophy the color remains, for the most part, unchanged, but the organ is firmer and denser. When fatty degeneration predominates it is pale, yellowish, soft and flabby.

Excess of pigmentary deposits is a common feature in atrophy of the myocardium, where it constitutes pigmentary atrophy. A certain amount of pigmentary deposit is generally found around the nuclei after middle life, but this should be excluded. In atrophic pigmentation there is great excess of the deposit with actual shrinkage and induration of the organ. On section the vessels are seen to be tortuous and the tissues of a chocolate hue.

On microscopic examination in simple atrophy, the muscular fibres are found to have become smaller and the muscular cells reduced in size with loss of their cylindrical shape, with a tendency to become fusiform. There may be an increase rather than a diminution in the transverse striation and longitudinal fibrillæ.

The pericardial fluid may be increased or diminished, usually the latter.

Symptoms.—The symptoms of atrophy of the heart are without distinctive features. They are those of heart weakness and constitute a part of the general clinical manifestations of the condition to which they are secondary. Feebleness, early exhaustion, breathlessness with palpitation and præcordial distress from pulmonary stasis, scanty urinary secretion with sediment and high specific gravity from renal congestion, attacks of syncope, ringing in the ears, spots before the eyes and mental confusion from defective blood-supply to the brain, are the usual symptoms. When these are present to a decided degree in maladies which are liable to waste the substance of the heart muscle, they may be considered as presumptive evidences of the presence of cardiac atrophy.

PHYSICAL SIGNS.—*Inspection* usually shows diminution or absence of the impact of the apex-beat, but when the patients are thin and the lungs are retracted, especially the left, there may be an exaggeration of distinctness with displacement of the impulse to the left of normal.

Palpation adds nothing except to confirm the signs of inspection. It should be borne in mind that in these conditions there is usually considerable emaciation which renders the movements of the heart more manifest to the sight and touch.

Percussion shows decrease of the normal area of dulness which is the most important and distinctive sign of cardiac atrophy. But error may arise in case of pulmonary emphysema which causes the area of cardiac dulness to appear much diminished.

Auscultation may disclose weakness of the heart-sounds, but if the heart is close to the surface, as it not infrequently is, they may appear intensified. When the heart muscle has become much weakened the first sound is muffled or absent and the second distinct. This clearness of the second sound is often observable in marasmic conditions and in pulmonary tuberculosis.

Diagnosis.—The recognition of the presence of the affection depends upon the diminution of the area of cardiac dulness. If this sign is determined, together with association with some wasting disease, general weakness, dyspnœa and feeble pulse, it is sufficient evidence to warrant the diagnosis of atrophy.

Prognosis.—The outlook depends entirely upon the primary lesion. In acute conditions, such as typhoid fever, it is favorable, but in chronic wasting diseases it is hopeless.

Treatment.—All efforts should be directed toward the primary malady and to sustaining the general strength.

FATTY INFILTRATION OF THE HEART.

SYNONYMS.—*Fatty Heart, Fatty Overgrowth, Cor Adiposum, Lipomatosis of the Heart, Obesitas Cordis.*

Fatty infiltration signifies an excessive accumulation of fat around the heart and in those localities where, in the healthy organ, a certain amount of adipose is normally present, together with an infiltration of the intermuscular fibres with fat and encroachment of the same upon the muscular structures reaching sometimes as far as the sub-endocardial tissues. Strictly speaking, it is not a degeneration, but inasmuch as it frequently leads to structural changes it is usually discussed under that head.

Ætiology.—The chief causes are the same as those which lead to the excessive accumulation of fat in other parts of the body. In the majority of instances the condition is associated with general obesity, the tendency to which depends either upon heredity or individual idiosyncrasy.

Apart from general obesity, chronic alcoholism is one of the most frequent causes. The condition is also occasionally observed in the aged, the insane and in certain cachectic conditions, as in carcinomata and tuberculosis.

Fatty infiltration belongs to after middle life and, according to some observers, occurs without regard to sex, while others claim that it is more common among men. It is never seen in infancy. It is not infrequently acquired by excess in eating and drinking combined with habits of indolence and lack of exercise.

Excess of fat, sugar and starch in the food are generally supposed to tend to obesity. This is doubtless true, but the quantity of food is also an important factor. Malt liquors especially favor this condition. On the other hand, stoutness may occur in persons who are not given to excesses in any way.

The underlying factor in all these conditions is defective metabolism which in some unknown way leads to the excessive formation of fat. The probable seat of the morbid process is the liver. Either more fat is formed than the economy can consume or the process of its elimination is defective. However this may be, there is no satisfactory explanation, especially when one takes into consideration the differences between individuals subjected to the same influences and habits of living.

Morbid Anatomy.—The deposition of fat between the grooves of the auricles and ventricles, and between the two ventricles is much in excess of normal. From these localities the adipose tissue passes around and envelop the ventricles to a greater or less degree, especially the right ventricle. In some cases it may measure an inch or more in thickness and hide the whole heart. As a rule, however, the overgrowth is not so general or so dense, and patches of the normal heart may appear through the fatty tissue. In extreme cases the process may encroach upon the muscular fibres, spreading among the intermuscular substance as far as the sub-endocardial tissues. This constitutes infiltration. Sometimes a column of fat runs through the heart walls.

When infiltration continues it leads to degeneration of the muscular fibres and dilatation. In cachexias fatty overgrowth and fatty degeneration may exist together. The association of arterio-sclerosis in this connection has long been recognized. It furnishes the cause of cerebral apoplexy which occurs not infrequently in this condition.

Symptoms.—Clinical phenomena arising from fatty heart, it is generally conceded, are confined to those cases where the accumulation of adipose tissue is accompanied by fatty infiltration of the muscular fibres. It is not probable that the mere presence of fat around the heart causes symptoms (Laennec, Corvisart).

Breathlessness on exertion, præcordial oppression, easily induced fatigue and sometimes giddiness and faintness are the symptoms which attend this condition. The dyspnœa is not paroxysmal, but appears only on exertion. In some instances it may be extreme, and not infrequently there is some œdema around the ankles. The digestive functions are good and the appetite in obesity cases is often excessive. The liver may show signs of enlargement and the stomach is often dilated. The pulse varies, sometimes being hard and full, at others, empty.

The condition is liable to progress; in advanced cases the signs of failing heart may appear. As a rule these cases progress slowly. In many death ensues from pulmonary œdema, while in others it occurs suddenly from syncope.

PHYSICAL SIGNS.—*Inspection* is negative except where there is true fatty infiltration with hypertrophy, in which case there may be some pulsations in the neck and diffusion of the præcordial impulse. *Palpation* shows feebleness of impulse. *Auscultation* reveals feebleness of the heart-sounds. The first sound is usually more affected than the second. Sometimes murmurs may be present, more especially systolic.

Diagnosis.—Excessive stoutness associated with the signs of general cardiac weakness forms the basis of diagnosis in cases of the obese type. In alcoholics and others, feeble heart action with dilatation points to its presence. A positive diagnosis, however, is often difficult.

Prognosis.—The outlook depends, to a great extent, upon the characteristics of each individual case. Hereditary tendency to obesity and habitual disinclination to exercise are unfavorable features. A determination on the part of the patient to reduce the accumulation of fat, and persistence and perseverance in carrying out plans directed toward that end, may be regarded as affording a good outlook. When infiltration is pronounced, especially if the patient is elderly, the prospect is unfavorable.

Treatment.—The general care of cases of fatty heart in obese persons is on the same plan as for the reduction of general obesity and corpulence. The diet and habits of life, therefore, are the chief features. The food should be such as will least tend to the formation of fat. Fluids should be used sparingly and alcohol generally prohibited and sugar and starch withdrawn. The quantity of food should also be diminished. The resistance exercises of Schott are of benefit, and are generally to be recommended so long as there is no reason to believe decided degeneration is present. General exercise should also be enjoined, mental as well as physical, the functions of the bowels and skin should be kept active. Bathing often proves beneficial.

Oertel's plan of treatment, which has given some good results, is as follows :

1. Reduction of the quantity of liquids taken at meals and during the intervals, the total each day being limited to thirty-six ounces. Frequent baths, including the Turkish bath, when not contraindicated. Promotion of diaphoresis.

2. Diet. Proteids form the chief part. Breakfast—a cup of coffee or tea with a little milk—about six ounces in all ; three ounces of bread. Noon—soup, four ounces, a little fish, roast beef, veal, game or poultry, salad or a light vegetable, in all about eight ounces ; bread or farinaceous pudding, one ounce ; fruit, six ounces ; no fluid at this meal, but in warm weather six ounces of light wine. Afternoon—tea or coffee, six ounces, with the same amount of milk or water ; sometimes one ounce of bread. Evening—one or two boiled eggs, one ounce of bread, a small bit of cheese, salad and fruit ; six to eight ounces of wine with four to five ounces of water.

3. Graduated exercises of various grades, a walk each day under constant supervision, and gradually increased as to distance and inclination. Acccording to Oertel, this is the most important part of the method, as it invigorates the heart muscle.

The presence of valvular lesions with insufficiency and broken compensation, and considerable atheroma of the arterial walls, are decided contraindications to the use of Oertel's method.

In cases of fatty heart arising from other causes there is no special line of treatment. In alcoholic cases the absolute prohibition of alcoholic stimulants, followed by the administration of strychnine and general tonics, with change of scene, hold out the only prospect of amelioration.

FATTY DEGENERATION OF THE HEART.

Fatty degeneration is the condition in which the muscular substance of the heart undergoes an actual metamorphosis and becomes fat. It is distinguished from fatty infiltration which is an encroachment of fatty tissue on the muscular structures and not primarily a change of substance; infiltration may, however, lead to real degeneration.

Ætiology.—It may occur at any age, but is very rare in infancy or youth. The period of life in which it has been most frequently observed is after forty, especially between the ages of sixty and seventy.

The affection is more prevalent among males than among females, and may appear in spare as well as in stout persons. Predisposition is observed in heredity and in sedentary habits. There can be no question but that some persons from inherited tendencies are prone to fatty degeneration of the heart, which in many instances accounts for the frequent occurrence of sudden dissolution in certain families. Habit of life is another important element. Lack of exercise, especially when combined with a hearty appetite and the use of alcohol, are conducive to these changes.

Whatever the cause the fundamental element in the ætiology of fatty degeneration is defective nutrition. The exciting causes are those which lie in the blood-supply, both as to quantity and quality, and those which arise from abnormal demands made upon the heart muscle. One of the most important factors in this wide and diversified ætiology is disease of the coronary arteries. In some instances atheroma of these vessels is followed by fibroid degeneration, while, in others, the changes are of a fatty character. Quain compared these conditions of the heart to those which take place in cerebral softening from similar causes, and has shown that the involved areas correspond with the atheroma of the coronary vessels.

Changes in the quality of the blood which may lead to fatty degeneration arise from both chronic and acute affections. The various forms of anæmia, especially leucocythæmia, progressive pernicious anæmia, chlorosis and inanition are often attended with fatty degeneration of the heart muscle. Repeated venesection in animals, it has been demonstrated, produces the condition. This accounts for its presence in persons who have suffered from long-continued hæmorrhages from any cause, such as hæmatemesis, hæmoptysis and metrorrhagia.

Fatty overgrowth, when it has reached the stage of infiltration and has penetrated the muscular structures, often proves the cause of degeneration of the muscular fibres and the two conditions are frequently found cöexistent.

Cachexias of all types are conducive to these changes. Thus, it may be observed in long-continued suppurative processes, tuberculosis and carcinomata. It not uncommonly appears in connection with diabetes and gout. In the latter instance it is probably caused by the diseased condition of the coronary vessels which so often results from the gouty diathesis. Bright's disease constitutes another factor, as in its latter stages it is liable to be attended with fatty degeneration of the heart muscle, thereby adding materially to the danger.

The acute affections which enter into its ætiology are numerous. It occurs in typhoid and typhus fevers, erysipelas, diphtheria, variola, septicæmia, pneumonia and malarial diseases of the tropics. In diphtheria the walls of the left ventricle and its musculi papillares are most likely to be involved, while the endocardium remains intact, except for the presence of some ecchymoses. In these cases the degeneration must be regarded as the direct result of the toxines in the circulation acting upon the heart structures. The same changes have been observed in connection with scurvy, purpura and hæmophilia.

Certain agents introduced into the system from without affect the heart to a marked degree, of these alcohol is the most common, though by no means the most powerful. Fatty degeneration of the heart is frequent among excessive drinkers and when sudden death occurs in these cases it is often attributable to its presence. The form of alcoholism most liable to affect the heart in this way is habitual and long-continued tippling, rather than violent, periodical excesses. As in the instance of hypertrophy beer drinkers are especially susceptible to this change. Phosphorus is the most quickly acting agent in producing fatty degeneration of the heart muscle. The process may begin a few days after it has been taken. Arsenic and oxalic acid poisoning act in the same way, but less speedily.

The effects of increased tension upon the heart muscle constitute a very important and comprehensive class of causal factors. Here will be observed the results of muscular strain from hard work or any other cause arising from excessive and long-continued physical exertion, the effect of high living, and the changes in the myocardium incident to failing compensation and fibroid degeneration. The influences underlying these conditions which are obviously manifold and of great variety have been previously discussed. In this instance, however, they must be regarded from the standpoint of their effect upon the heart muscle rather than as to their relation to changes in the valve structures. They are operative in every grade of life, more especially in the two opposing conditions of wealth and poverty. In the former it is the high living, in the latter the continuous physical exertion which leads to the high arterial tension and consequent morbid changes in which fatty degeneration often plays such an important rôle. Thus these two extremes may bring about a similar result. The same condition is not infrequently observed among gymnasts, oarsmen and bicyclists.

Conditions of excessive muscular hypertrophy from any cause are frequently found associated with more or less fatty degeneration and fibroid changes in the heart walls. In severe forms of inflammation of the pericardium and endocardium microscopic examination often reveals the presence of fatty changes. Again, the presence of growths, especially of gummata, may develop fatty degeneration.

Finally, in some cases, no cause can be found and the origin of the lesion must be entirely presumptive, depending upon probable mal-assimilation in the nutrition of the heart muscle or upon some unrecognized abnormal state of the blood itself.

Morbid Anatomy.—The degenerative processes may be either localized or diffuse. The appearance of the heart varies considerably according to the extent of the changes. In the strict sense of the word, fatty degeneration in itself does not give rise to increase in the bulk of the organ, but on account of its common association with dilatation and hypertrophy enlargement generally accompanies its presence. Again, diffuse degeneration may lead to dilatation, while the muscular tissues, on the other hand, in dilatation and hypertrophy, eventually undergo fatty change. Thus the process may be the result as well as the cause of dilatation. In some instances the heart may be found to become actually diminished in size. This may occur in association with protracted chronic affections.

The degenerative changes may involve the entire organ, but are usually more pronounced in the left ventricle, especially near the apex, which is first implicated. The septum is the next part affected, and last, and to a lesser degree, the right ventricle. According to Quain, in about one-half of the cases both ventricles are involved, but that when only one is the seat of the lesion it is the left twice as frequently as the right. The auricles are little affected, but occasionally suffer.

The heart as seen with the unaided eye, after the removal of the overlying fat which is sometimes abundant, shows loss of the normal redness of the muscle, which is brownish-yellow or yellowish, according to whether the changes are acute or chronic. This condition is usually more localized in the neighborhood of the apex. Patches of fatty deposit may be observed scattered over the surface of the muscle apparently superficially, but on microscopical examination it will be found that they are deep-seated, involving the musculi papillares, which have a streaked appearance, and the tissues of the trabeculæ. In these localities, in advanced conditions, the deposits of fat may be seen with the naked eye under the surface of the endocardium.

In cases arising in connection with chronic diseases the color of the heart muscle is pale, frequently with areas where the paleness is intensified. When there is local obstruction with consequent stoppage of the blood-supply there may be a patch of circumscribed pallor.

In acute cases the tissues may be dark from blood stains, with sometimes occasional points of loss of color. The entire organ is liable to be affected, although the left side of the heart shows more decided alterations than the right. In extreme cases the patches of changed tissue may resemble an abscess.

Microscopic examination reveals the most positive evidences of fatty degeneration. The process consists of a gradual displacement of the muscular elements by fatty granules. The first noticeable change is the presence of fine, minute granules within the muscular fibres near the nuclei, lying longitudinally and generally parallel to the fibrillæ. There are few other changes early, the structures at this period being little affected. When the condition has advanced there is increase in the number of the granules, with loss of striation and disappearance of the nuclei in the affected fibres. The granular condition of the degenerated fibres presents a cloudy appearance which may be mistaken for cloudy swelling or albuminoid degeneration. A distinction may be made by applying acetic acid, which dissolves albuminoid granules, brings out the granules of fatty degeneration in strong relief and dispels the cloudiness. Staining with osmic acid will also develop characteristic features in fatty degeneration.

Under a more powerful lense the microscope shows the granules to be oil globules; with them may be seen dark bodies which are called pigment granules. This gives a darker color to the fibre. As the condition advances the muscular tissue is more or less displaced by the oil globules, and its consistency eventually changed to a pale, yellowish, flabby and easily broken substance, which imparts a sensation of greasiness to the touch. Sometimes excess of pigment may cause a mottled appearance. In conjunction with these changes, the muscular fibres may become affected in a singular manner. They may become, as it were, disjointed, that is, there are intervals between the fibres instead of firm and continuous connection. This condition is termed fragmentation. What influence it may have in contributing to failure of the heart powers it is not possible to say, as the chief factor must be the displacement of the active muscular fibres by fat.

The association of fatty degeneration of the heart muscle with other affections of the myocardium has been the subject of frequent allusion in the discussion of the respective conditions with which it is liable to occur. In fibroid degeneration, fatty infiltration, dilatation, hypertrophy, atheroma, thrombosis and in some cases of peri- and endocarditis it appears as a retrogressive process, the product of other and distinct pathological conditions.

In connection with the fatty changes in the heart, fatty degeneration may be found in the liver, tunica intima of the arteries and epithelia of the kidneys. The vessels of the brain may also become the seat of fatty changes, sometimes without evidence of similar changes in other arteries.

The exact nature of the metamorphic process is unknown. Fat exists in chemical combination in all animal tissues. The amount in the heart is from 2 to 3 per cent. of the entire weight of the organ. To say that the transformation of muscle tissue into fat is a chemical process does not throw much light upon the subject.

Symptoms.—Much vagueness surrounds the clinical manifestations of fatty degeneration of the heart. This is due, in part, to the fact that a considerable degree of fatty degeneration may be present without giving rise to symptoms, and in part to the association of the lesion with other lesions, either as a cause or, more frequently, as a resulting secondary retrogressive process, which are themselves attended with disturbances of the circulation.

As in other forms of chronic cardiac disease, the development of symptoms largely depends upon the habits and circumstances of the patient, that is, upon the demands made upon the heart. The more even and quiet the mode of living, and the greater the freedom from worry and anxiety, the greater the chances of immunity from symptoms. There are, however, many exceptions where symptoms develop, notwithstanding solicitous care.

The course of the affection displays decided diversity. The onset may be slow and gradual or it may appear suddenly under the influence of some apparently slight cause. Sometimes a severe strain may stir into activity a condition which otherwise might have remained latent. The same may be said of emotional influences. The reserve power of the heart, however, is such that a considerable fatty change may be tolerated before symptoms make their appearance. In those forms arising in connection with dilatation from any cause, and other retrogressive changes of the myocardium, the progress is slow and the condition may continue for a long period without exciting other than a moderate degree of disturbance; there will always be, however, a liability of more pronounced and serious symptoms manifesting themselves with more or less abruptness. In cases which originate from anæmias and acute diseases the progress of the degeneration is rapid.

When the disease has progressed sufficiently to give rise to symptoms, there is often implication of the coronary arteries or hypertrophy with or without dilatation.

The symptoms generally resemble those of dilatation. The patient usually first suffers from breathlessness, especially on exertion, and with attacks of palpitation. He also complains of debility and loss of muscular power. From being active he appears indolent and sluggish. The general condition of the system points to a lowering, the temperature in some instances being below normal. There may be often a sensation of distress in the præcordial region. Sometimes this may amount to actual pain which radiates upward and outward to the shoulders and arms. Attacks of angina pectoris may appear, due, doubtless, to disease of the coronary arteries, rather than to the fatty degeneration itself.

The general appearance is in no way characteristic. The countenance may be either pale or it may wear the signs of apparent health. In chronic alcoholics with fatty degeneration of the heart there is frequently marked pallor. In the aged, yellowness of the conjunctiva from the presence of fatty tissue, and the arcus senilis may be present. It is deemed, however, by many that this is of no positive diagnostic importance. It may,on the other hand, be regarded as of value in connection with other signs. Sometimes there may be some œdema of the lower extremities, but this symptom may be confined, for the most part, to the later stages with excessive dilatation.

The pulse reveals changes which vary according to the type of the process and the presence of associated affections. In cases arising from anæmias and acute diseases the arterial walls are soft and elastic; in those connected with chronic affections, especially in the aged, they may be hard and unyielding. The vessel is usually observed to be somewhat empty with diminution of blood-pressure. The rate shows decided variation, but generally it is accelerated, being about a hundred or more per minute. In some chronic forms, on the other hand, there is decided slowness, as low as thirty or forty per minute.

The rhythm may also suffer changes. Irregularity and regularity are both common. There may be loss of the pulse-wave in the vessels; that is, every pulsation of the heart may not be observed at the radial artery.

Cerebral symptoms are liable to appear at an early date as a natural consequence of the diminution of blood supplied to the brain. Vertigo, faintness with slight nausea and momentary loss of mental power are common. These distressing sensations usually quickly disappear, especially upon the exertion of will power. Loss of mental force often proves a very marked feature. The patient is no longer able to apply his mind or display his former fortitude. At times there appears to be a sort of mental vacancy of which he is fully and painfully conscious. The memory becomes defective and often the disposition irritable. A tendency to somnolence is a feature. Even after a long sleep the patient complains of drowsiness. On the other hand, however, sometimes insomnia may prove troublesome, due, undoubtedly in many instances, to sleeping during the day, a tendency to which the patient is often unable to overcome.

In pronounced forms of the affection there may be sudden attacks of blindness and actual syncope ; the latter may appear in several forms. Generally the patient lies, semi-unconscious, with pale face, cold surface and extremities and very slow pulse. In more unusual instances the syncopal attacks may assume the characteristics of pseudo-apoplexy as observed in fibroid degeneration. This condition is remarkable in that it is not distinguishable for the time from the true apoplexy due to cerebral hæmorrhage. The patient lies on his back entirely unconscious. In some cases these attacks may resemble epilepsy. The convulsions, however, are not as severe as in epilepsy, but unconsciousness is complete and the pulse is very slow, sometimes only twenty beats per minute. It would appear that the cause of the convulsive condition is due to the defective circulation of the brain from the feeble heart action.

The respiratory symptoms which appear early become intensified as the degeneration advances. The early dyspnœa on exertion becomes more persistent and paroxysms of cardiac asthma of great intensity may occur.

In some instances, during sleep, Cheyne-Stokes respiration will be observed. This phenomenon, to which attention was first called by Stokes and which was afterwards more fully described by Cheyne, consists of a peculiar rhythm of respiration in which, having attained a maximum degree of intensity, the respirations with even graduation of diminution grow less and less until they finally cease, then there is a pause, after which they begin with faint efforts, which gradually increase in intensity in the reverse of the manner in which they declined. This continues until the maximum is reached; the phenomenon is then again repeated. Cheyne-Stokes respiration is by no means pathognomonic of fatty degeneration of the heart, as it may be observed in many conditions in which there is defective blood-supply to the medulla oblongata. It is an unfavorable sign. Other changes in the respiration may be noted in the form of more or less constant tendency to sighing and yawning.

The alimentary tract shows evidence of impairment in loss of appetite, feeble digestion and general torpor of the bowels and liver; the latter often is enlarged. The muscular layers of the stomach may lose their tone and allow of distention from the gases of fermentation. This condition may sometimes exert a deleterious influence by pressure upon the heart from upward expansion, especially when the diaphragm is relaxed. The line of gastric resonance may often be traced horizontally from the base of the ensiform cartilage to the normal position of the apex in the fifth interspace. The pressure of the distended stomach on the weakened heart may be sufficient to arrest its action and cause sudden death.

Renal disturbances are also common in the advanced stages, the urine may be scanty, albuminous and contain casts. In the latter stages, when dilatation has become excessive, the signs of stasis of the circulation develop. The pulse grows more feeble and arrhythmical and dropsical symptoms may appear, the latter, however, in uncomplicated fatty degeneration, is never well marked. In most instances, whatever the cause, fatty degeneration terminates with greater or less rapidity in failure of the heart differing in no way from heart failure from other causes.

While the course of the lesion is slow, it may suddenly terminate by arrest of the heart from asystole. Sudden death in aortic regurgitation is most always associated with the fatty degeneration. A rare contingency is rupture of the heart walls which may occur from slight causes.

PHYSICAL SIGNS.—It is only when the chambers of the heart become enlarged that physical signs can be appreciated, as fatty degeneration of mild degree affords no physical evidence of its presence.

Inspection and *palpation* shows feebleness of the apex impulse, which usually occupies a position near normal. Stokes pointed out that sometimes the only impulse perceptible is that felt at the lower end of the sternum at its right border. *Percussion* may afford no sign of enlargement except in cases associated with hypertrophy. *Auscultation* reveals enfeeblement of the normal sounds, the first is usually shortened and less pronounced. In some instances it may be inaudible at the apex where the second sound alone may be heard. In many cases a weakened first sound may be heard at the lower end of the sternum. The interval between the first and second sounds is sometimes shortened, though this is usually normal. Murmurs may be heard in conditions of hypertrophy and associated valvular disease, febrile disturbance and anæmia, otherwise they will be absent.

Diagnosis.—The absence of characteristic and positive physical signs renders the recognition of fatty degeneration of the heart difficult. In many instances the diagnosis must be inferential rather than positive.

When in a given case symptoms indicative of a lowering of the circulation are present, especially with attacks of angina, or with apoplectiform or epileptiform seizures, and at the same time when there is absence of the physical signs of murmurs or marked changes in the heart-sounds, or of disturbance in the relations of the left and right side of the heart, the condition may be regarded as pointing decidedly to the presence of fatty degeneration of the heart. Again, certain features connected with the pulse will prove of diagnostic assistance. A weak, unsustained pulse may be observed in many chronic conditions. It may indicate simply debility, or something more. In weakness of the pulse arising from organic disease, when the patient is made to walk quickly, if the heart is fatty the pulse will become irregular and shorter and much more feeble; whereas, in the absence of organic disease, it will, for the time, increase in volume and remain regular.

In valvular disease, dilatation and hypertrophy, fatty degeneration may be regarded as probable when enfeeblement of the heart action is more than that which is deemed proportionate to the valvular defect or the amount of dilatation.

The presence of a reasonable and valid cause will also greatly assist the diagnosis. The history of the case and the age of the patient must likewise be taken into consideration.

Sudden death in old alcoholics in whom there have been no signs of valvular disease points to fatty degeneration, likewise persons of full habit who gradually develop the symptoms of heart weakness may be considered as sufferers from fatty degenerative changes of the myocardium.

In acute diseases and anæmias failure of the heart may generally be regarded as evidence of fatty degeneration of its walls.

The fact that the disease is so frequently an associated or secondary affection renders it difficult and often impossible to separate it from other conditions. Uncomplicated forms of the lesion resemble advanced dilatation in many respects, but the latter may be distinguished by the presence of the greater prominence of dropsy, of an increase in the area of dulness, and by the absence of the peculiar attacks simulating apoplexy and epilepsy.

From fibroid degeneration it is often impossible to make any distinction, as the same clinical manifestations, including the angenoid and cerebral symptoms, may be present in both conditions. *Vide* Fibroid Degeneration.

Prognosis.—An opinion as to the future in a case of fatty degeneration of the heart rests, to a very great extent, upon the origin of the changes. When arising in connection with acute diseases there is always great danger, at the same time strong hope for ultimate, complete recovery. In protracted fevers the condition must be always regarded as grave. Recovery depends mainly upon the strength and vitality of the patient. It is also largely influenced by the period of invasion of the degenerative process. As a rule, the later its appearance in the course of an acute affection, the more favorable the outlook.

In the instance of fatty degeneration arising from phosphorus, arsenical or oxalic acid poisoning the course of the affection is rapid and unfavorable. The same may be said when it is associated with cachexias, tuberculosis, Bright's disease, and pernicious anæmia. In cases dependent upon other forms of anæmia when the primary cause can be removed the situation is favorable. In those associated with fatty overgrowth and infiltration there are very reasonable grounds for belief that treatment would prove helpful if diligently pursued, but the continued self-denial and perseverance so essential are often lacking, and the situation is corespondingly unfavorable. In cases due to alcohol, much also can be accomplished if its use is discontinued, but in confirmed alcoholics this is impossible unless the patient is placed in a sanitarium.

In fatty degeneration associated with dilatation and other structural changes of slow process, the patient may live for several years, but the outlook for ultimate restoration is hopeless. Senile types which form a part of a general decay of the arterial system are not necessarily always unfavorable; if not excessive they may exert, according to Sir William Jenner, a conservative influence upon the circulation of the cerebral vessels by lowering arterial tension, as otherwise the hypertrophied heart beating with full force might easily break down the walls of vessels weakened by atheroma.

When death occurs suddenly in persons suffering from fatty degeneration of the heart it usually follows some indiscretion or profound emotional influence, especially the former. A run for a train, a long, fatiguing walk, especially if against the wind, may overtax the weakened muscle and lead to its arrest. Sometimes death may be instantaneous, sometimes it may be preceded by attacks of syncope with a more gradual decline of the heart power. Again, indiscretion in diet in persons troubled with gaseous fermentation may, as mentioned, lead to sudden dissolution by pressure on the heart. In most of these cases there will be reason to believe that with care the patient might have lived much longer.

To resume, in brief it may be said that generally the prognosis of fatty degeneration dependent upon acute disease is grave as far as the immediate danger to life is concerned, but if the acute condition is passed safely there is usually ultimate restoration of the heart muscle. In toxic conditions it is bad; in chronic conditions, as far as immediate danger is concerned, it is fair, but hopeless as to recovery.

Treatment.—It is evident from the diversity of its origin that the treatment must present several aspects.

In acute fatty degeneration of the heart, strychnine stands first, digitalis and the remedies of its class with alcoholic diffusible stimulants are also indicated. Inhalations of oxygen may prove of great value, and succeed when other means have failed.

Cases arising from toxic influences depend entirely upon the withdrawal of the poison, after which treatment may be pursued according to the individual indications.

In those forms of the lesion due to infiltration, overgrowth and chronic conditions, regulation of the diet, which should be plain and nutritious, rest, both mental and physical, with complete freedom from worry and gentle and carefully supervised exercise, should be enjoined. Frequently the treatment of baths and graduated exercises as pursued at Nauheim, Germany, is attended with excellent results. For obese persons a course at Marienbad, Bohemia, is recommended.

The indications for remedies in chronic forms are on the same general lines as for fibroid degeneration and cardiac dilatation. Nitroglycerin is the foremost remedy. Evidences of peripheral resistance in the circulation and angenoid pains especially demand its use. The iodides, in small doses three times a day long continued, also frequently prove of benefit. Iodide of sodium or iodide of strontium may be administered in place of the potassium. Iodide of arsenic is also of undoubted value, especially when there is anæmia, the second decimal trituration, every two or three hours, may be given. Chloride of gold and soda should also be borne in mind. Other important remedies are iodine and phosphorus.

When there are evidences of cardiac insufficiency, with dyspnœa, weak, irregular pulse and œdema, the general line of treatment discussed under failing compensation will be indicated. Here digitalis may be used with good results. It is often most effective when employed in association with nitroglycerin.

The other remedies of the digitalis group may also prove of service. Strychnine should also be remembered as one of the most effective drugs for spurring on a flagging heart, but the use of remedies of this class should generally be confined to a limited period and to emergencies. Under no condition should they be administered without due consideration, for it should always be borne in mind that in the weakened condition of the vessels overstimulation is dangerous.

FIBROID DEGENERATION OF THE HEART.

SYNONYMS.—*Chronic Myocarditis, Cardiac Sclerosis, Cardiac Cirrhosis, Fibro-Myocarditis, and Sclerotic Myocarditis.*

The diversity of terms which is here presented is significant of the variety of opinions which are entertained in regard to the morbid processes which take place in the muscular substance of the heart. Several distinct forms of degenerative changes may be said to be included under the head of chronic myocarditis.

Ætiology.—The initial factors in the causation of fibroid degeneration are changes in the quality of the blood and changes in the circulation itself, that is, the amount of blood circulating through the heart and consequent alteration in the blood-pressure.

The morbid conditions which are operative in the first place are alcoholic excess, abuse of tobacco, gout, rheumatism, diabetes, syphilis, lead poisoning, and chronic nephritis; in short, all conditions which interfere with the tissue metamorphoses and which tend to promote arterio-sclerosis. These agents usually first induce sclerosis of the coronary arteries which is secondarily followed by fibroid degeneration of the heart muscle.

Rheumatism causes chronic myocardial inflammation by extension of the inflammatory process of the peri- and endocardium to the heart muscle itself. Especially is this likely to happen when the pericarditis is attended with adhesion. Typhoid fever may form the starting-point of the lesion which may follow as a sequela. Syphilis may act in two ways: first, by producing gummata, and, secondly, by endarteritis it may cause defective nutrition.

In cases which originate from alterations in the blood-current through the heart, where the changes in the quality of the blood are secondary, it will be found that valvular deficiencies are the most fertile causes. Under these conditions the heart walls are subjected to a constant increased tension from the backward pressure, at the same time the blood is more or less stagnant or impure. As a result the heart muscle undergoes degenerative changes in the form of chronic induration.

Chronic nephritis, emphysema and atheroma may result in the same condition by obstructing the circulation.

Another potent cause is over-strain from laborious and arduous occupations. The effect produced is similar to that of valvular disease. The increased tension of the blood-pressure causes degeneration of the heart walls. Thus, certain vocations, such as that of porters, workers in foundries, shovellers, etc., are prone to induce fibroid degeneration of the heart. Likewise persons indulging in excess in rowing, bicycling, running or any athletic sports requiring great muscular effort, may suffer in the same way.

The part of the coronary circulation in the causation of fibroid degeneration is of the utmost importance. The causal factors of the disease may early affect these structures and thus diminish the blood-supply to the heart muscle. The disease of the coronary vessels leads to the formation of thrombosis and subsequently to anæmic infarcts. Embolic processes may also arise from vegetations within the heart cavities. Thrombosis of a coronary vessel is necessarily a matter of the utmost gravity, as it is inevitably followed by serious anatomical changes; sometimes by sudden death.

Fibroid degeneration or chronic myocarditis is essentially a disease of after life. It is more frequently observed among males than females, as might be inferred from its ætiology.

Morbid Anatomy.—The parts of the heart most frequently implicated are the left ventricle at the apex, the inferior portion of the ventricular septum, and the papillary muscles. There is enlargement from dilatation with more or less hypertrophy, but the walls are thinner than they should be considering the increase in bulk. The diseased processes may be either diffuse or circumscribed. In the first instance the excess of connective tissue often leads to enlargement of the organ.

The diseased areas consist of grayish or grayish-yellow patches which vary in size from microscopical points to one or two inches in diameter. The yellow hue is due to the presence of fatty degeneration. Sometimes there are blood-stained points from thrombotic deposits. There is generally induration, but in some instances there may be softening of the tissues.

When the structural alterations are of a strictly inflammatory type there may be a blending of the interstitial and parenchymatous changes, but usually there is a predominance of one element over the other. When the parenchyma is the principal seat the muscular changes are general. This type is more frequently observed as the sequela of rheumatism or specific fevers. According to Virchow the changes here are a softening of the tissues. The muscular fibres lose their striations and become filled with albuminous granules and later by fat.

The changes in the heart walls may lead to disarrangement of the valvular and orifice structures with characteristic signs and phenomena. Similar effects may result from the implication of the musculi papillares. Compensatory hypertrophy of the uninvolved portion of the heart may arise, which, in some cases, will account for the presence of hypertrophy with fibroid degeneration, but in many others it is the direct consequence of the sclerotic degeneration.

The coronary arteries are early involved in the inflammatory and degenerative changes. Sometimes these consist of an endarteritis deformans with or without calcareous degeneration, sometimes an endarteritis obliterans. The former is more common and is often associated with nephritic disease and fibrous degeneration elsewhere. The tendency of these vascular changes is encroachment on the calibre of the coronary vessels, and consequent faulty and perverted nutrition of the myocardial structures. Another very important feature in these morbid alterations of the coronary circulation is the liability to thrombosis and embolism, the result of which is anæmic infarcts leading to subsequent sclerosis in the heart muscle.

Microscopic examination of the tissues shows hyperplasia of the interfibrillar connective tissue and subsequent fibrous degeneration. In turn the muscle exhibits various stages of degeneration, granular and fatty. Atrophy of the muscular tissue follows as a result of the compression due to the fibroid degeneration.

When an infarct occurs as a consequence of thrombosis in a sclerosed vessel, the appearance of a point of lodgment varies according to the time of observation. If seen early there is paleness only, while if seen later the point is soft and yellowish. The microscope shows the fibres disintegrated and the striæ presenting a hyaline appearance, while surrounding the seat of the infarct there is fibrous proliferation.

Symptoms.—The clinical manifestations of fibroid degeneration strongly resemble those of fatty. While in most instances they are decided, invariably so when the degenerative processes are pronounced, they are, on the other hand, frequently characterized by irregularities and indefiniteness. In some instances post-mortem has revealed the presence of indurations of the myocardium when during life there was absence of symptoms. In such cases hypertrophy was completely compensatory.

The symptoms which first attract attention are dyspnœa, palpitation and præcordial distress ; the latter is often described by the patient either as a sense of weight or of constriction. There also may be throbbing of the arteries, flashes of light before the eyes and tinnitus aurium. Vertigo and faintness may prove a source of annoyance. Pain around the region of the heart is often present in varying degrees. In some instance it occurs in the form of paroxysms of true angina with all the concomitant symptoms of that condition. Occasionally, recurrent anginal attacks, with or without arrhythmia, are the only phenomena which may manifest themselves in chronic myocarditis. In such cases the condition is to be attributed to the sclerosis of the coronary arteries. Breathlessness is generally one of the prominent symptoms, and sometimes during sleep there may be Cheyne-Stokes respiration. Bronchial catarrh is often observed and cough is not infrequent.

The urine is usually diminished and of high specific gravity, and often contains casts. Nervous symptoms in the form of insomnia are common. Sometimes there are evidences of loss of mental power.

In the latter stage there is general progressive debility, while fatigue on slight exertion is attended with breathlessness and palpitation is easily excited. The appearance of the patient does not present anything essentially characteristic. In some instances, however, close scrutiny may detect some tortuosity of the temporal arteries, at the same time there may be pulsation of the carotids, either one or both.

The pulse-rate shows decided changes. It may be slow, full, intermittent and irregular, that is, there may be bradycardia and arrhythmia. Occasionally, it is accelerated, especially as the heart fails. Slowness, however, is common, the pulse being fifty or even forty per minute. Reduction of pulse-rate (bradycardia) with intermittency (arrhythmia) and angina are features strongly suggestive of chronic myocardial inflammation.

Pseudo-apoplexy may occur. The patient may be suddenly seized with vertigo and unconsciousness, with slowness of the pulse (Adams-Stokes syndrome). Frequently this condition comes on after a heavy meal or some imprudence in diet. As a rule, the period of unconsciousness is brief and is not attended with paralysis. In some instances, on the other hand, slight convulsive movements of an epileptiform character or temporary paralysis may follow, which may continue for a few hours. Attacks of this kind may occur at various intervals. There is always more or less danger that they lead to true apoplexy from cerebral hæmorrhage or leave some evidences of loss of mental vigor.

During the early period of the affection gastric and secondary circulatory disturbances may be absent, but when the lesion has advanced, relative insufficiency of the mitral valve followed by the same condition of the tricuspid arises with all the visceral and circulatory changes which develop in association with failure of the right side of the heart. Hence, dropsy, engorgement of the abdominal organs, cardiac asthma and pulmonary œdema appear in the last periods of the disease.

In some instances the case may not go on to the development of secondary heart symptoms, but may terminate suddenly in death.

Physical Signs.—*Inspection* does not, as a rule, reveal any notable features connected with the præcordium. The radial arteries frequently show the evidence of atheroma.

Palpation discloses feebleness of the apex-beat with displacement downward and to the left from the dilatation and hypertrophy.

Percussion furnishes evidences of increase in the area of dulness.

Auscultation gives a general muffling and indistinctness of the heart-sounds or the systole may be strong. The aortic second sound may be either accentuated or double. The mitral sound is loud, and with dilatation there may be a systolic murmur due to relative insufficiency. Tricuspid insufficiency may also be recognized. With the development of insufficiency the heart action may be very rapid.

Diagnosis.—The lack of individuality in the symptoms renders it impossible to speak with assurance in regard to the recognition of chronic myocarditis. The signs of failing heart, however, with the phenomena of valvular disease in connection with the physical signs point to fibroid degeneration. This is equally true of fatty degeneration, and unless the history of the case can throw some light on the situation the diagnosis in many instances must be somewhat a matter of conjecture.

The history of previous attacks of acute gout or latent forms of the affection as manifested by eczematous eruptions, recurrent gastro-intestinal disorders, cystic irritation, insomnia, mental depression and irritability, in connection with the symptoms discussed, point to the presence of fibroid degeneration dependent upon gout.

The affections which resemble fibroid degeneration are valvular disease of the heart, fatty degeneration and fatty overgrowth.

Valvular lesions may be disregarded during the period before the chronic myocardial process has developed a murmur. When a murmur has made its appearance it may be due either to the fact that the papillary muscles and tendinous chords have been involved or that relative insufficiency has become established. In such instances the clinical course of the diseased conditions presents the same features as in other forms of valvular deficiencies, and there is no definite way of recognizing the presence of fibroid degeneration except by such assistance as may be afforded by the clinical history.

The differentiation between fatty and fibroid degeneration of the heart is difficult and, in some cases, impossible. There can be no doubt that the latter condition is more frequently associated with arterio-sclerosis, yet fatty degeneration often occurs with hypertrophy dependent upon sclerosis and fibroid degeneration. While both may present the same general symptoms, the presence of attacks of syncope, arterio-sclerosis, strong cardiac systole and accentuation of the aortic second sound suggest the probability of fibroid disease. Arcus senilis, when present, is regarded by some observers as a sign of fatty degeneration.

Fatty overgrowth is chiefly distinguished by the presence of obesity and by the habits of life, being found, for the most part, among heavy beer drinkers and those given to over-eating and drinking combined with lack of exercise. Vertigo may be present, but it is usually slight. Attacks of true syncope are rare, while bronchitis, asthma and emphysema are common.

Prognosis.—The outlook is serious, and when its presence is suspected opinion must always be guarded. While the tendency of the affection is toward a fatal issue, a considerable degree of myocardial change is not incompatible with long life.

With such opposing aspects, in order to form an opinion as to the future, the previous history of each case must be carefully investigated. This constitutes a very important element in prognosis. If a considerable period has elapsed since the condition has developed, and the cardiac impulse and the first sound are strong, the outlook may be regarded as not unfavorable. Again, if syphilis is the cause there is reason to suppose that under the use of antisyphilitic treatment the process may be removed to a degree, if not wholly.

The effect of muscular exertion, for example, going up-stairs, should be noted. If there is much breathlessness it signifies considerable damage to the myocardium. Angenoid pain and attacks of syncope are unfavorable to a marked degree. The association of arterio-sclerosis, chronic interstitial nephritis and diabetes mellitus must be regarded as affording a gloomy outlook.

The overshadowing possibility of sudden death must also be taken into consideration, as it not infrequently occurs. Nevertheless, with care and treatment much can be done and life greatly prolonged.

Treatment.—In treating a patient suffering from a chronic myocardial inflammatory process, his whole course of life, habits, exercise and diet should receive careful attention. Rest to the feeble heart-muscle must be ordered, and at the same time a certain amount of judicious exercise may be allowed in certain cases.

Metabolism should be encouraged in every way. The free use of diluents and gentle general massage are useful. In cases which are not advanced the baths of Nauheim and the resistance exercises of Schott may be beneficial.

The diet should be nutritious and light. It should be arranged according to any diathetic conditions which may be present, especially gout. Patients suffering from the latter, together with those in whom there are evidences of arterio-sclerosis or nephritis, often are benefited by a course at Carlsbad, Marienbad, Vichy or Kissengen abroad, or at Saratoga, Bedford Springs and the Hot Springs of Arkansas, in this country.

Among drugs, potassium iodide in small doses, long continued, are unquestionably beneficial. Other remedies of decided value are arsenicum album, arsenicum iodide, mercurius iodide, chloride of gold and soda, baryta carbonica, baryta iodide, iodide of soda and the iodide of strontium.

Angenoid attacks and pain should be treated along the general line of angina pectoris. Arsenicum iodide, nitroglycerin, cold or hot applications to the præcordium, nitrite of amyl, and morphia, hypodermically, are the most satisfactory remedies. The continued, but cautious, use of nitroglycerin may prove useful in preventing attacks, of this nature, the remedy being especially indicated in cases associated with arterio-sclerosis and high arterial tension. For the throbbing sensation in the arteries, and insomnia, bromide of soda in 10-grain doses, about four times in twenty-four hours, is often helpful.

When the heart begins to fail it will be necessary to resort to cardiac stimulants and tonics, notably strychnine, in doses of $\frac{1}{80}$ to $\frac{1}{60}$ grain three times a day. Spartein and caffein should also be considered. Digitalis may also be used with benefit when there are signs of cardiac insufficiency with weak, irregular, rapid pulse, dyspnœa and œdema.

When there is arrhythmia with slow pulse and tendency to syncope, generally without dropsy or marked dilatation, digitalis is not indicated. The remedies which, on the other hand, should then be considered, are stimulants, such as alcohol and ammonia; morphia, if there is extreme restlessness and anxiety; and nitroglycerin, if there is a hard pulse.

AMYLOID, GRANULAR, HYALINE AND CALCAREOUS DEGENERATION AND BROWN ATROPHY.

The following conditions, while constituting important pathological changes, do not occur as distinct clinical entities.

Amyloid or Albuminoid Degeneration.

This condition is rare. As a rule, the changes are confined to the blood-vessels and connective tissue between the fibres, the muscular fibres generally escape. Milky opacities of the endocardium of the right auricle are common, although any of the heart chambers may show evidences of amyloid deposits. Response to the usual stains may be obtained, but rarely can the condition be detected except by the microscope. The causes are the same as those of amyloid degeneration of other viscera.

Cloudy Swelling, or Granular Degeneration.

This simple form of degeneration, which is one of the most frequent, attacks the heart as it does other muscular structures. While not a fatty change, it is thought by many pathologists that it may constitute the initial step in that process. It occurs in the course of acute infections, such as diphtheria, septicæmia, scarlet fever, typhoid fever, erysipelas, etc., and in hyperpyrexia, Bright's disease, and in any protracted, exhausting illness. It may also be observed in acute inflammations of the serous membranes of the heart, where it may appear as the result of the inflammatory process or arise from the same causal influences. Whatever its exciting cause, the active factors are doubtless the presence of toxines in the blood acting upon the protoplasm.

Microscopic examination of the heart shows the organ to be softened, somewhat enlarged, œdematous to the touch and on section of a cloudy, grayish color, in marked contrast to the red of the normal heart. In advanced cases rigor mortis may not be prominent, nor will it be entirely absent.

The microscope shows swelling and distention of the muscular cells which are filled with granular matter, while the nuclei are less clear than normal. The cell contents are albuminous and not fatty ; the granules clear up under acetic acid and remain insoluble in ether and alcohol.

Hyaline Degeneration.

Hyaline degeneration occurs sometimes as the result of protracted fevers (hyaline transformation of Zenker). The substance of the muscle appears to undergo coagulation, the fibres are swollen, translucent and homogenous with loss of the striæ. The process resembles vitreous changes in other muscles. The fibres may become fragmented, the separation taking place at the junction of the muscle cells or directly through the cell wall. For this condition Renaut proposed the name of segmentary myocarditis. Sometimes it is observed with amyloid degeneration. The heart to the unaided eye has a clouded appearance.

Calcareous Degeneration.

This form of degenerative process consists of a deposit in the fibres of the heart muscle of carbonate and phosphate of lime and of the salts of magnesium. It is generally found in senile tissues, especially in the intima and media of the vessels. In these conditions the calcareous infiltration is usually preceded by fibrous and atheromatous degeneration, and when these changes take place there is reason to suppose that a disturbance of the circulation of the part precedes the process. It may also be observed in connective tissue which develops as a result of irritation, as, for example, in chronic inflammation of the pericardium. More frequently calcareous infiltration occurs as the sequela of the inspissation of the contents of pus cavities which arise in connection with myocardial abscess or purulent myocarditis. The nature of the process is, for the most part, hypothetical, but as lime salts are present in the blood and lymph, it is supposed that in some unknown way they become isolated and in this form infiltrate the muscular tissues.

Brown Atrophy.

Pigmentary atrophy or brown atrophy is a degenerative condition of the heart muscle in which there is a deposition of pigment in association with an increase of firmness and diminution in the size of the organ. The pigment is of a yellowish-brown or a dark red-brown color. Its source is not precisely known. Microscopically it is seen to collect chiefly around the nuclei of the muscular fibres and between the formative cells where it obscures their structure. The condition occurs in old age, cachexias, marasmus and other wasting conditions. It may sometimes also be observed in certain forms of valvular disease.

ANEURYSM OF THE HEART.

Aneurysm of the heart is a sacculated dilatation of a portion of the heart wall. The older writers applied the term to enlargement resulting from hypertrophy and dilatation. The lesion is commonly described as acute or false and chronic or true aneurysm. The former is the result of acute inflammation of the endo- and myocardium, the latter, of slow degenerative changes in the heart wall. Aneurysm of the valves belongs properly to affections of those structures and is discussed under that subject.

Ætiology.—In the acute varieties the aneurysmal dilatation arises from a sudden giving way of a weakened area in the myocardium. The most frequent causes are acute endocarditis and myocarditis, especially when these processes are of the ulcerative variety. In both instances the rapid inflammatory changes which tend to softening and ulceration give rise to conditions favorable to rupture and extravasation of blood in the heart walls. When myocarditis is purulent, rupture of an abscess may form the cavity of the aneurysm.

Sometimes the lesion rises from occlusion of the coronary artery from embolism, which leads to rapid softening and thus forms the source of the aneurysmal dilatation.

In true or chronic aneurysm the development is a gradual dilatation of a weakened part. Although it is not impossible to suppose that an acute ulceration or rupture may be the starting-point, the most frequent cause is fibroid change or chronic myocarditis. The wide range of influences which underlie this condition are thus causally related to cardiac aneurysm. One of the most active factors is obstruction of the coronary circulation, the change being arterio-sclerosis followed by thrombosis. Thus atheroma in its various manifestations, including that of the aorta near the mouth of the coronary vessels, must be taken into account. Endarteritis obliterans must also be considered.

The relation of syphilis to aneurysm of the heart is most important. According to Walshe, it is responsible for the majority of cases. A certain proportion find their origin in fatty degeneration where a circumscribed spot in the walls gives way without complete rupture. Tuberculous growths may also give rise to the condition, and, in rare instances, chronic endocarditis. A still more unusual source of origin is pericardial adhesions.

It is claimed that strain, in which there is partial rupture of the walls, may be the starting-point of cardiac aneurysm ; while this is doubtless probable it can only be possible when the myocardium has previously undergone some degeneration.

The affection is more prevalent among males, and after forty years of age. This is probably due to the fact that increased arterial tension from muscular exercise is operative to a greater extent among men, and that the effects of its influence is not apparent until middle life. Cases are on record, however, where the affection has been found in young children.

Morbid Anatomy.—The essential feature in aneurysm of the heart is change in a part of the myocardium, whereby its power to resist the pressure of the blood within the heart chambers is diminished. The lesion may be single or multiple, but it is usually the former. In size it varies all the way from a fraction of an inch to that of a cocoanut (Quain). In rare instances it may give no external evidences, either because it is so small or because it burrows within the walls of the heart, in other words, is a dissecting aneurysm. The heart in the latter, and very rare condition, is usually hypertrophied and dilated.

In acute cases the seat of the aneurysm is generally in the left ventricle near the apex. In rare instances it may appear in the upper part of the left ventricular septum ; when in the latter locality it may protrude into the right ventricle, but if developed during fœtal life the reverse obtains. When situated near the base it may grow upward near the aorta. Pericarditis is usually attendant upon such aneurysmal developments. Acute aneurysms usually either discharge their contents into the heart chamber, thereby giving rise to embolism, or rupture.

Chronic aneurysm is preceded by extensive changes in the myocardium. Its seat is the same as in the acute forms. Its walls are thin. In the early stages of formation they are composed of normal muscular fibre and more or less fibroid tissue. As the lesion progresses and the cavity expands the muscular element decreases, while the fibroid increases until, in some cases, a membrane only intervenes between the endo- and pericardium. The latter generally endure for a while unless they have been weakened by previous ulceration, or when rupture is the source of the lesion. When aneurysm reaches the surface of the heart there is thinning of the pericardium.

The interior of the aneurysmal sac varies greatly. Some are saucer-like depressions which neither cause obstruction nor deposition of coagula, while others are decidedly sacculated. In the latter there is more or less constriction at the opening into the heart chamber. When the opening is of good size it is usually irregular, when small it is generally more even. The internal surface is smooth except when the aneurysm is due to rupture, in which case it is rough. In the sacculated varieties there is always a tendency to the formation of coagula which are sometimes deposited in layers, but in many cases non-laminated blood clots are found.

Upon the external surface there is usually adhesions and thickening of the pericardium. In a case of aneurysm of the right auricle reported by Berthold there was erosion of the ribs and the appearance externally of a pulsating tumor.*

Symptoms and Diagnosis.—Aneurysm of the heart is devoid of characteristic signs or symptoms. Its presence is seldom recognized during life. The symptoms to which it has been observed to give rise are dyspnœa, palpitation, præcordial distress, feeble pulse and enlargement of the area of cardiac dulness.

Certain features have been pointed out as characteristic, for example, a tendency of the patient to bend forward so that the chest almost touches the knees (Bucquoy), and the presence of a pulsating prominence of the chest (Anders), but they cannot be said to be of any practical aid to diagnosis.

Prognosis.—The outlook is decidedly unfavorable, as there is a possibility of further expansion of the sac and rupture of its walls which in some cases is the cause of death, but the more frequent termination is heart failure.

Treatment.—The aspect of the subject leaves little to be said as to treatment which must be on general principles according to the symptoms of each case.

DISEASE OF THE CORONARY ARTERIES.

The profound influence which disease of the coronary arteries exerts in the causation of cardiac affections renders it desirable to consider the subject individually. It is generally stated that the coronary arteries are terminal vessels, but it is a matter of some doubt whether this is literally true, inasmuch as a fluid injected into one finds its way into the other. All agree, however, that the anastomosis is unequal to the maintenance of a collateral circulation sufficient to nourish a given area after obstruction of the artery which supplies it.

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* Pepper, System of Medicine, Philadelphia, 1896.

If the theory of Huchard* is accepted, coronary disease may be classified as functional or paroxysmal, and permanent, or organic.

The functional form is characterized by a spasmodic contraction of the walls of the vessels which cuts off or limits the normal blood-flow. The cause of this phenomenon is attributed by Huchard to the influence of nicotine poisoning upon the muscular coating of the arteries. It is manifested by attacks of angina and is confined to those who smoke in excess. It is not of frequent occurrence.

Permanent or organic forms are due to organic lesions and may be classified as follows: Arterio-sclerosis, endarteritis obliterans, aneurysm, occlusion, thrombosis and embolism.

(1) **Arterio-Sclerosis.**—This form of degeneration, also known as atheroma, and endarteritis deformans of Virchow, may attack the coronary arteries either as a natural accompaniment of similar changes in the aorta or of a general implication of the arterial system, or it may occur independently of either of these two conditions. In seeking its causation it may be found as part of the general process of degeneration which attends old age and as the result of gout, syphilis, nephritis, strain from hard labor and chronic lead poisoning.

It is essentially a disease of middle and advanced life and is more frequent among males than females. Heredity plays an important part, especially in premature decay of the arterial system. Syphilitic disease of the coronary arteries in this connection need not be mentioned as a separate entity, as it is essentially a condition of atheroma. One or both arteries may be affected.

The first change is the appearance on the inner surface of the intima of flat, rounded elevations, which gradually blend with the surrounding tissues. These thickened areas are more transparent, paler and softer than normal. Their surfaces may be either smooth or somewhat wrinkled. There are also similar points which are yellowish or white and rougher, and others which are yellow with decided thickening. When these changes are extreme, the inner wall of the vessels is more or less thickened, uneven, and the vessel itself dilated. As the process advances the intima loses its shining appearance and roughness, erosions and ulcerations develop.

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* Traité Clinique des Maladies du Cœur et des Vessaux, Henri Huchard, Paris, 1893.

An extensive amount of fatty degeneration occurs. This is followed by a collection of yellowish débris and softening. The term atheroma has been applied to this stage of the process (ἀθήρη = gruel, ὄμα = tumor). The collections sometimes may rupture and thus give rise to an ulcerating surface. Subsequently there follow deposits of lime salts in the form of thin calcareous plates in the walls. These may encircle the whole circumference of the vessel and render it like a cord or tube. With calcification there may be some tortuosity.

The effect of these changes is diminution in the calibre of the vessel and loss of elasticity and contractile power. Calcification, if extensive, may encroach on the lumen of the vessel to such an extent as to lead to complete occlusion.

There are no symptoms or signs which belong individually to the disease, but the changes which subsequently occur in the substance of the heart are of the most profound character, being those of fatty and fibroid degeneration, aneurysm of the walls, thrombosis and embolism of the coronary arteries.

(2) **Endarteritis Obliterans.**—This is another variety of endarteritis which occurs in the coronary artery, more especially in the smaller branches, and is a result of syphilis. Its pathological changes are chiefly those of thickening of the intima of the vessel to such a degree that its lumen is entirely obliterated. The process may involve one side or the entire circumference of the vessel.

Microscopic examination shows proliferation of connective tissue rich in cells, with complete absence of fat or calcareous deposits. When the occlusion is not absolute the endothelium of the lining membrane of the vessel will be present. The outer coats of the artery remain intact as the changes are confined to the inner lining.

The disease is followed by the development of chronic myocarditis with the subsequent changes which characterize that lesion.

(3) **Aneurysm.**—Any part of the vessel may be involved. The most frequent cause is atheroma, in rare instances, embolism. The size of the aneurysm is generally about that of a pea, though it may be much larger. There are no symptoms which definitely de-

note its presence. Sudden death from rupture of the aneurysmal sac is the usual termination. The lesion is uncommon.

(4) **Occlusion.**—Occlusion may occur from obstruction by a calcified plate closing the arteries or from the encroachment of the sclerosed and thickened walls. The seat is usually at the orifice of the artery, more rarely in the main trunk or one of its branches.

(5) **Thrombosis.**—This is a common result of disease of the lining of the vessels. It arises chiefly from atheroma, but is likewise caused by acute and chronic endarteritis near the orifice of the artery, and possibly by acute inflammation. Thrombotic vegetation on the aortic valve has been known to occlude one of the coronary arteries.

The result of thrombosis of either the main trunk or of the intermuscular branches of the coronary arteries may be instant death, on the other hand, the patient may live. In the latter instance the result is infarction of the area supplied by the affected artery. Infarction may arise as the result of both thrombosis and embolism, but inasmuch as it is more frequently the product of the former it is described in this connection. The most frequent seat of the process is in the left ventricular septum and anterior wall of the left ventricle. This is due to the fact that the descending or anterior interventricular branches of the left coronary artery are more often the seat of sclerotic degeneration.

The size of the infarct corresponds, in the main, to the area supplied by the occluded artery, but it may be only a part. It projects above the surface and is irregular in shape, but rarely wedge-shaped like infarctions elsewhere.

Both pale, or anæmic, and hæmorrhagic infarcts occur; the former are the more common. Newly formed anæmic infarcts are firm, opaque and yellowish-white in the centre, surrounded with a border of hyperæmic and hæmorrhagic infiltration. Under the microscope they show coagulation necrosis, fibres without nuclei, indistinctly striated and brittle from sclerosis. The central portion may undergo softening and form a concretion known as myomalacia cordis. The infarct may reach as far as the endocardium, where it may cause a parietal thrombotic process, or it may extend to the pericardium where it sets up inflammation. In some instances a favorable reaction may set in which leads to absorption, or the process may be followed by fatty or fibroid degeneration or parietal aneurysm.

(6) **Embolism.**—This may arise from disease of the arterial coats, also from valvular disease, pyæmia and other infections. Metastatic abscesses in the heart in pyæmia are not uncommon.

The lodgment of emboli in the coronary arteries and their branches is attended with the same consequences as in the case of thrombi, but the latter is of much more frequent occurrence. The sequelæ are the same in both instances, sudden death or profound præcordial distress, dyspnœa, delirium, coma and symptoms of collapse followed by dissolution; in case of survival, infarction, fatty and fibroid degeneration, or aneurysm.

THROMBOSIS OF THE HEART.

Thrombosis of the heart is the formation of a coagulum or thrombus within its cavities during life.

Ætiology.—Thrombosis is caused by lesions of the heart walls, changes in the blood and retardation of the circulation; the latter is an active contributing cause and not alone sufficient, as an abnormal condition of the endocardium, such as inflammation, atheroma, calcification and bacteria is necessary.

A thrombus or clot in the heart of the living subject is a product of the formation of fibrin, the separation of which from the blood is attributed to the contact of a fibrin ferment with the fibrinogen of the liquor sanguinis. The ferment appears in the blood as the result of dissolution of the leucocytes by toxines. In all lesions of the endocardium there results a collection of white blood-corpuscles at the diseased areas. These become changed into finely granular fibrin.

In the process of disintegration of the white blood-corpuscles the fibrin which they contain becomes liberated. It then unites with the fibrinogen and forms a thrombus. The morbid conditions of the heart walls and blood which favor the development of thrombi occur in both acute and chronic diseases, especially in those characterized by the presence of blood poisons and retardation of the circulation and weakness of the heart. Among the most important acute affections are endocarditis in connection with rheumatism, diphtheria, typhoid and other infectious fevers, lobar pneumonia, myocarditis and all infectious and septico-pyæmic diseases. In chronic lesions it is observed with valvular disease, secondary dilatation, Bright's disease and long-standing affections, such as carcinomata, tuberculosis and marasmus.

Morbid Anatomy.—True thrombi are generally situated in the sacculated appendage of the auricle, at the apices of the ventricles and behind the columnæ carneæ, as far removed as possible from the blood-current. They are more frequent in the right ventricle and in the auricular appendages, less so in the apex. They may sometimes be observed as excrescences projecting into adjoining cavities, as from the left ventricle into the aorta. They are adherent to the endocardial surface, sometimes requiring considerable effort to detach them. Their formation may be sudden or gradual.

Cardiac thrombi are divided by Hertz into four classes: (1)* Small bodies adherent to the heart wall with softened centres, suggesting the appearance of cysts. These are the globular vegetations of Laennec. (2) Large masses which may fill a considerable section of the heart, especially the appendices of the auricle. (3) Pedunculated thrombi. (4) Masses without any adhesion floating free in the blood. The latter are the ball thrombi of Recklinghausen, and are most frequently observed as free bodies in the right auricle in association with mitral stenosis. Thrombi are also described as parietal and occlusive, the former being flattened deposits lying against the walls, and the latter, as the term implies, acting as obstructions to the circulation. In size they vary all the way from a mustard seed to a bird's egg. They occur singly or in groups and are composed largely of fibrin with red and white blood-corpuscles in varying proportions. Generally they are grayish-yellow in color, but are also sometimes red or white.

Jahn classified thrombi according to color, as red, white or mixed. The red contains a preponderance of red blood-corpuscles, the white an excess of the white blood-corpuscles, and the mixed, both red and white in varying proportions. The difference in color is attributed to their method of origin. The red are the result of rapid coagulation, the white are caused by a constant deposition of flowing blood, the mixed represent a combination of both and are usually white at the beginning.

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* Quoted by Whittaker, Twentieth Century Practice, vol. iv., New York, 1895.

Variations in color depend not only upon the amount of red blood-corpuscles present, but also upon the age of the clot and the chemical change it has undergone. Old clots are generally grayish or yellowish in hue.

On section, thrombi, in many instances, are found to be laminated and of a yellowish-gray color, with patches of red and black. The centre contains a sanious, foul, puriform fluid. Some are firm and friable, others show calcareous changes. Sometimes old clots are covered by a layer of recent formation. Microscopically they are found to consist of degenerated cells and detritus.

Detached fragments of thrombi may be carried into the circulation, lodge in the visceral or peripheral tissues and set up embolic processes.

Symptoms.—The clinical manifestations of cardiac thrombosis, while extremely grave, are not in any way typical, being those of failing heart power. There is great dyspnœa, præcordial distress, irregular, weak and rapid pulse, cyanosis, syncope, restlessness and jactitation. There may also be convulsions, delirium and coma. The patient seldom lives longer than the third day, sometimes only a few hours and, in some instances, death is instantaneous. The symptoms may appear suddenly or gradually according to the rapidity of the formation of the thrombus and its situation in the heart. In the first instance they may be ushered in with great intensity and terminate in death immediately or after a brief interval. On the other hand, when of slow formation, they may be of less abrupt onset and of milder intensity. In some instances again the coagulum may not only be of slow formation, but also may be of small size and so placed that it does not interfere to a great extent with the blood-current. Under these conditions death does not necessarily occur immediately. Gradually, however, the clot becomes larger and the serious symptoms develop.

When the thrombus occludes a valvular orifice or an efferent vessel, death is immediate, or within a few hours. The breaking up of a thrombus is attended with the lodgment of emboli in other parts of the organism. As the right side of the heart is the most frequent seat of the thrombotic process, the possibility of pulmonary embolism must be borne in mind.

Physical Signs.—These are, feeble impulse and arrhythmia. There may be increased area of dulness and the sudden development of a bruit, but in most cases the heart-sounds evince no signs beyond extreme feebleness.

Diagnosis.—Clinically, there are no definite data upon which to establish a positive diagnosis. If the patient survive the formation of a thrombus, as he may do in the instance of those of slow formation, and there afterwards appear embolic deposits elsewhere, the presence of cardiac thrombosis may be regarded as more than presumptive.

It is important to differentiate between the clots of ante- and post-mortem origin found on necropsy. The ante-mortem clots, called agony clots by the older writers, are those formed during the death agony, or soon after, and have no pathological signification. They are distinguished from clots formed during life by their large size, position and consistence. They may be red, white or yellow, semi-translucent and succulent. They are more common and are of firmest consistence in persons who have died of acute inflammatory diseases. They may adhere quite firmly to the trabeculæ of the heart or may be free; they may also extend from the heart cavities into the vessels. The largest are found in the right auricle.

Prognosis.—This is unfavorable in all cases, not only on account of the thrombosis, but also on account of the primary disease. In most instances death occurs within a few hours or, at the most, in a few days. Exceptions to the fatal outlook are found in those cases where the thrombi are small and have changed to vegetations and where they become organized and thicken the heart wall.

Treatment.—Treatment must be according to symptoms, to sustain the heart and relieve the distress as far as possible.

EMBOLISM OF THE HEART.

Embolism of the heart is a detached fragment of a thrombus, which has been formed elsewhere in the circulatory system, and which has found lodgment in one of the cavities of the heart. It may prove the starting-point of a thrombus.

Clinically, cardiac emboli may give rise to symptoms similar to those of thrombosis, which are not distinguishable from the same, or they may be a cause of sudden death in the course of some primary disease.

The presence of cardiac embolism may be suspected when sudden death or grave symptoms of cardiac failure appear in the course of diseases predisposing to the formation of thrombi.

SPONTANEOUS RUPTURE OF THE HEART.

Spontaneous rupture of the heart is an uncommon and, it is needless to say, an exceedingly grave lesion. It occurs only when the myocardium has become enfeebled by disease and never when the heart is normal.

Ætiology.—In all cases of spontaneous rupture examination has disclosed the presence of a degenerative process, and in many instances, in addition, an exciting cause has been recognized. The former which alone renders the latter possible includes the various degenerations to which the organ is subject. The most common cause is disease of the coronary arteries in which the lumen of the vessels suffers contraction with consequent impoverishment of the nutrition of the myocardium, softening of the muscle, aneurysm or necrosis and abscess from thrombotic and embolic deposits. Fatty degeneration is another potent causal factor. Many cases are also attributable to fibroid degeneration. In rare instances new growths and parasites in the walls give rise to the accident. Rupture of the papillary muscles and trabeculæ, to which allusion has been made in the discussion of valvular disease, may be caused by similar influences and is sometimes called partial rupture, but the term spontaneous rupture as here presented implies a complete rent in the myocardium.

Exciting causes are strain, muscular effort or strong emotions. The greater proportion of the cases have occurred after lifting heavy weights, climbing a hill or carrying a heavy burden. Cases are also on record which have resulted from the effects of violent coughing, vomiting, straining at stool and sexual intercourse. Dulaux reported a case which was induced by tetanic convulsions. Hudson, one by the efforts of parturition, and Tissot one by epilepsy. Violent emotional influences, such as a fright, excessive joy or anger have been followed by spontaneous rupture in conditions of advanced disease of the heart walls.

Morbid Anatomy.—The principal seat of rupture is the anterior wall of the left ventricle. According to Odriozola, in 33 cases out of 71 it was near the apex, in 28 near the middle portion, and in 10 near the base. Rupture posteriorly is rare, while the left border and apex seem to be free from this accident. Rupture of the right ventricle and the auricles may occur, but is very rare; out of 132 cases Odriozola says that the left ventricle was the seat of lesion in 96 instances, the right in 22, the right auricle in 10, the left in 2, and the left auriculo-ventricular sulcus in 2.* In the greater proportion of cases the tear consisted of a single opening, in a few only were there more than one. In size it has been found to vary from a small slit to one an inch and a half long. In shape it is variable, straight, curved, or irregular. Sometimes its inner and outer opening do not correspond.

On opening the pericardium more or less blood is found, although the sac is rarely distended. Sometimes the blood is deposited in one or more layers over the surface. Where there are several layers the outer is composed of loose, soft coagula, while the innermost are dense, firm and whitish. In some cases there are ecchymotic deposits beneath the pericardium. After the removal of the clots and coagula the blood is usually seen oozing from the open wound. Sometimes it is difficult to find the inner opening. The entire absence of blood has been reported.

The gross general appearance of the heart in spontaneous rupture varies according to the nature of the primary pathological process. It is commonly paler than normal, with numerous red patches from hæmorrhages into the tissues.

Microscopic examination shows the presence of atrophic lesions, especially those of a pigmentary or granular character. In many cases endarteritis deformans and obliterans with thrombosis is observed. Valvular lesions are common, and in some cases affections of the pericardium are also present.

Symptoms.—Rupture of the heart is usually attended with symptoms of such a severe and rapidly fatal character that nothing definitely diagnostic is obtainable. Death is frequently instantaneous. In some instances the patient is seized with an agonizing pain in the præcordial region similar to that of angina, and like it, radiating to the shoulders and arms. At the same time there is dyspnœa, cyanosis, cold perspiration, pallor, weak and irregular pulse, and all the indications of speedy collapse. Sometimes there is nausea and vomiting and occasionally convulsions. Frequently the patient becomes unconscious and dies in that condition. While either an instantaneous or speedy death occurs in the majority of instances, life may sometimes be prolonged for several hours or even several days. Under these circumstances there are grave symptoms of præcordial distress with dyspnœa, cyanosis and extreme weakness of the heart. In some cases there is absence of pain.

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* Quoted from Gibson, Diseases of the Heart and Aorta, Edinburgh and London, 1898.

PHYSICAL SIGNS.—There are no distinctive signs. When examination is possible the heart is observed to be very feeble, with its sounds muffled or imperceptible. The area of dulness may be increased.

Diagnosis.—The diagnosis of spontaneous rupture of the heart is difficult and frequently impossible. In patients of advanced life in whom the existence of disease of the coronary arteries, fatty degeneration or chronic myocardial inflammation has been previously recognized, the sudden onset of violent pain in the chest with dyspnœa, tumultuous action of the heart, cyanosis and collapse followed by fatal syncope, may be regarded as suggestive of rupture. When death is instantaneous even presumptive evidence is lacking, but sometimes the previous history of the case may point to such a contingency.

In cases where life is prolonged, rupture of the heart wall may present some points of similarity to angina. The conditions may be distinguished from the fact that angina is usually characterized by a tense, regular pulse and by the history of repeated attacks, while in rupture the pulse is very feeble and irregular, and there is never a history of a previous similar attack. In rupture also the area of cardiac dulness may increase.

Prognosis.—The nature of the lesion permits of little comment as to prognosis. Cases are on record where life was prolonged two, three, four, and even twelve days. The delay of the fatal termination under these circumstances was found to be due, in some instances, to the presence of a thrombus in the wound.

Treatment.—Generally there is no time to employ remedial measures. In those cases where life is not immediately terminated ice should be applied to the præcordium to quiet the tumultuous action of the heart, while heat and sinapisms should be applied to the extremities. Stimulants, such as alcohol, strychnine, camphor and digitalis, should be administered. For the angenoid pain, morphia sulphate, hypodermically, and inhalations of amyl nitrite may be employed.

SYPHILIS OF THE HEART.

Modern investigation has shown that syphilis of the heart is not as infrequent as was formerly supposed. Any part of the organ may be affected, muscular structure, membranes, vessels or nerves.

Ætiology.—Syphilis of the myocardium is an affection of the tertiary stage. It occurs as early as one and as late as eighteen years after infection. The average is ten years. It is more frequent among males than females, and those in middle life. According to Jullien hereditary syphilis also affects the heart.*

Morbid Anatomy.—The usual manifestation is the gumma, but a sclerotic myocarditis and endocarditis also occurs. In the former the mass varies in size from a pea to a pigeon's egg. Its most frequent seat is the left ventricle, more rarely the right ventricle, the auricles and septum. The gumma may be completely hidden in the muscular structures, so as to be visible only on section, or it may project from the surface outwards or inwards. The largest tumors are found in the septum from which they may protrude into either or both cavities. Cardiac gummata are of a pale gray color and of the same general appearance and consistence as similar deposits elsewhere. They may be either multiple or single. When situated near the membranes they excite inflammation. They may remain quiescent or, after a period, may soften or break down completely and form abscesses which discharge their contents into the pericardiac sac or into the heart cavities.

Syphilitic myocarditis generally occurs in association with gummata with which fibroid degeneration is commonly associated. It is also found independently as a sclerotic myocarditis. The process may be either circumscribed or disseminated, usually the latter. The changes are fibroid in character, accompanied with the development of cicatricial tissue and atrophy. Patches of fibroid induration with endarteritis obliterans are found. Softening may arise which results in aneurysmal dilatation of the heart walls. There is also often some inflammation of the peri- and endocardium. The influence of syphilis in the production of aneurysm of the heart has long been recognized and has been discussed under that subject.

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* Traité Practique des Maladies Veneriennes, Louis Jullien, M.D., Paris, 1879.

Syphilitic endocarditis may be either valvular or parietal. According to Mraček it appears in the second stage. It results in valvular defects which follow cicatricial contractions. Scattered throughout medical literature are reports of cases where the various valves have become affected. Fœtal syphilitic endocarditis has been described by Forster.

When attacking the heart syphilis has an especial affinity for locating in the coronary arteries, and more or less evidence of syphilitic degeneration is found in these vessels in all forms of cardiac syphilis. A syphilitic endarteritis is frequently the starting-point of other forms of profound disturbances and changes, such as aneurysm, angina pectoris, myocarditis or any of the several forms of degeneration to which the heart walls are subject. The possibility of syphilitic neurosis of the heart has been established by the report of cases of atheroma and kindred affections disappearing under the influence of anti-syphilitic treatment.

Symptoms.—Clinically, syphilis of the heart may fail to present any evidence of disturbance, or it may, on the other hand, be the cause of most serious symptoms. In either case its onset is insidious. In the latent varieties sudden death may be the first intimation of trouble with the heart which is demonstrated only by post-mortem examination. In those types which are attended with signs, the symptoms do not differ materially from those of fibroid degeneration. In advanced stages there is dyspnœa which, according to Jullien, is a prominent symptom, venous engorgement, œdema about the ankles, full pulse, enlargement of the heart, and feebleness of the sounds. Some cases are anginal in character and the patient suffers with severe attacks of angina pectoris.

Diagnosis.—The recognition of syphilis of the heart is surrounded with difficulties. The lapse of time since the primary affection and the frequent absence of all specific symptoms for a long period tend to lend obscurity to the situation. Gummata may remain incysted and give no evidence of their presence for an indefinite period when suddenly the heart begins to fail. In those cases attended with evidences of heart weakness or angina, if there are indications of constitutional infection or the history of the same, the diagnosis may be established, especially if there be amelioration of the condition under the administration of specific treatment.

The presence of osteocopic pains, alopecia, obscure cutaneous affections, or any of the many manifestations of tertiary syphilis in association with symptoms which point to myocarditis, will warrant the assertion of the specific origin of the latter.

Prognosis.—The outlook in syphilis of the heart is bad. Its latency and the impossibility of its early recognition are unfavorable features. Sudden death is very common; on the other hand, cases are on record which have been apparently cured. If it were possible to recognize the condition earlier the outlook would obviously be much more favorable.

Treatment.—The treatment is manifestly along the line of specific medication in association with such cardiac remedies as may be called for by the condition of the individual case. Iodide of potash is the principal drug. It may be combined advantageously with digitalis. Mercury is also effective. When these two cardinal remedies fail, aurum should be tried.

TUBERCULOSIS OF THE HEART.

The heart muscle is more rarely affected by tuberculosis than its membranes. In acute miliary tuberculosis it is not uncommon to find gray tubercular deposits in the myocardium. Sometimes, though rarely, large cheesy nodules are found at varying depths in the muscular structure, beneath the pericardium, the latter being often implicated in conjunction with other organs and tissues of the body.

Tuberculous myocarditis may occur with tuberculosis of the peri- or endocardium.

There are no definite symptoms by which tuberculosis of the heart can be recognized during life.

NEOPLASMS OF THE HEART.

Tumors of the heart are very rare and usually secondary. Carcinoma, sarcoma, myoma, fibroma and lipoma may all occur.

Carcinoma.—Cancer is the most frequent of cardiac neoplasms. In the greater proportion of cases it is secondary, there being but seven primary cases on record. Bodenheim in 1877 collected forty-five cases, cardiac cancer, to which others have been added. Köhler, Tranchon, Willigk, each mention a number of autopsies, the sum total of which is 21.954; of this number twenty-one showed cancerous disease of the heart.*

The affection occurs at any age, but is more frequent in middle life and among males. It is probable that cases occurring in infancy and youth are sarcoma. It is found in both ventricles and auricles, either as a single deposit or disseminated throughout the heart structure. In the cases of Bodenheim,† seven were in the left ventricle, three in the right ventricle, two in the left auricle; the remainder were multiple. The connective tissue between the muscular fibres seems to be the favorite point for the process to first appear.

In most cases cancer of the heart is the result of extension by contiguity from cancer of the mediastinal glands, œsophagus, pleuræ and lungs. In rare instances it may arise by metastasis from the disease in distant organs. Clinically, there is lack of definite features. The disease may remain entirely latent or may be attended with grave cardiac symptoms. In those cases in which it was found on post-mortem examination there had been præcordial distress, pain, palpitation, dyspnœa, irregular pulse and weak heart action.

Sarcoma.—This form of malignant disease is even more rare than cancer. It may occur in various forms, lympho-sarcoma, melanotic, etc., and at any age, but is more common in earlier life than cancer. The left side of the heart is the most frequent seat of the growth. Cases have been reported by Gross, Jacobi, Roninschein and Redtenbacher. The symptoms when present do not differ materially from those of cancer.

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* Diseases of the Heart and Aorta, G. A. Gibson, M.D., Edinburgh and London, 1898.

† James T. Whittaker, M.D., Twentieth Century Practice, vol. iv., New York, 1895.

Myoma.—The few cases which are on record show the tumor to have been situated in the left auricle in the form of a nodular, transparent, gelatinous body attached to the wall by a thin pedicle. In all the cases the patients were subject to apoplectiform attacks which were attributed to embolism from detached fragments. The form of myoma known as rhabdomyoma may occur in the heart walls as a circumscribed, nodular mass. According to Hamilton cardiac myomata are generally congenital.

Fibroma.—A few cases have been found. Vulpian reported a case of fibromatous cyst with purulent contents in the left auricle which ruptured, causing typhoid symptoms and apoplexy. Virchow reported a case of congenital cancerous myxoma. A number of other cases are on record which were attended with emboli.

Tuberculous Tumors.—*Vide* Tuberculosis of the Heart.

PARASITES OF THE HEART.

Four varieties may invade the heart: the echinococcus, the cysticercus cellulosa, the actinomycis and the pentomastonum denticulatum. The first two are the most frequent. In some cases the parasites are scattered throughout the body, the heart sharing the general invasion; in others, there is but a single deposit.

Echinococcus growths may attain considerable size; they are generally multiple and secondary to similar growths in other organs. The growth varies in size from that of a pin head to that of an orange. Of the seven hundred cases of hydatid collected by Davaine and Cobbold,* twenty-five were of the heart and pulmonary artery. The right side of the heart seems to have been more frequently implicated. The cyst may be situated beneath the pericardium, beneath the endocardium or in the myocardium. In some instances the sac may swing from a pedicle free within the heart cavity. It may thus act as an obstructant or it may rupture.

Actinomyces were found by Paltauf in the walls of the left ventricle in a phthisical patient whose case had been diagnosed as tubercular pericarditis.†

Cysticerci have been found in the left ventricle of the heart.

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* A Treatise on Entozoa of Man and Animals, Thomas Spencer Cobbold, London, 1879.

† Twentieth Century Practice, vol. iv., New York, 1875.

The symptoms are those of impairment of the heart muscle and of the functions of the valves, and do not differ from those derangements when they arise from other causes.

An important feature is the liability of the tumors to soften and allow the parasites to become detached. The result of such a change is the formation of emboli and danger of sudden death.

The diagnosis of cardiac parasites can seldom be made. The recognition of deposits elsewhere in conjunction with symptoms of cardiac distress is suggestive, while not conclusive. The presence of emboli may also lead to a diagnosis. A tumor may reveal itself by pressure. Parasites may be detected by aspiration of tumors in the anterior mediastium and pericardium. Treatment is palliative.

SECTION V.

NEUROSES OF THE HEART.

ANGINA PECTORIS.

SYNONYMS.—*Sterno-cardia, Sternalgia, Neuralgia of the Heart, Syncope Anginosa.*

ANGINA pectoris is an affection characterized by paroxysms of agonizing pain in the præcordial region, chiefly substernal, which radiates down the arms, across the shoulders and to the back of the neck and occipital region.

In the strict sense of the word angina pectoris is not a disease, but a syndrome associated with organic and functional disturbances of the heart and aorta and dependent upon a complex variety of causes and morbid conditions which are neither constant nor always definite.

Walshe* divided the affection into true and false angina, the former including those varieties which arise in connection with organic diseases of the coronary arteries and aorta; the latter, those dependent upon neuroses, vaso-motor, reflex and hysterical disturbances. The dividing line between these forms is not always closely drawn, neither has the classification been universally recognized.

Ætiology.—TRUE ANGINA.—With rare exception the affection is associated with arterio-sclerosis of the coronary arteries. The various factors which lead to this process must therefore be considered as essential and component parts of its ætiology.

Predisposing influences are found in heredity, age, sex, occupation and station in life. Heredity plays no small part. The tend-

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A Practical Treatise on the Heart and Great Vessels, Walter Hayle Walshe, M.D., 4th ed., London, 1873.

ency in certain families to degenerative changes in the arterial system is well recognized, and in the instance of this affection is not infrequently observed. The case of the Arnold family has often been cited as a notable example. Matthew Arnold, his father, Thomas of Rugby, and his grandfather all died of angina pectoris.

In regard to age, comparatively few cases occur under forty, the majority being over fifty. Exceptions are observed in aortic disease, aortitis and aneurysm. Cases, however, are occasionally seen among the young, one of twelve years of age being on record. Males are more liable to the affection than females. It is evident that the influence of age and sex arises from the fact that arterio-sclerosis is for the most part confined to middle life, and that males are more exposed to the causal agencies.

Laborious occupation requiring long continued physical exertion is considered as conducive to angina pectoris, owing to its tendency to give rise to arterio-sclerosis from increased blood-pressure. While this is true in some instances it cannot be said that the affection is more frequent among the laboring classes, if one may judge from hospital patients. On the contrary, it is more often observed among the rich and well-to-do, and in men of intellectual pursuits. Prolonged, severe mental strain may exert a powerful influence on, if not actually give rise to, angina pectoris, by causing increased arterial tension. Probably for this reason, in part at least, the affection is more frequent among brain workers.

Foremost among diseases which underlie true angina pectoris is gout. There can be no question but that this is one of the most important factors in its ætiology. Diabetes is not an infrequent cause, and a certain proportion of cases has been found associated with it. Valvular disease of the heart is not commonly attended with true angina pectoris. Præcordial pains are frequent, but they are not true angina. Aortic disease, especially insufficiency, is the form of valvular lesion which is most likely to be associated with the affection. In mitral disease it is very rare.

Chronic interstitial myocarditis, probably more than any other disease, is the most constant source of angina. The sclerotic degeneration which typifies its presence may be either general or partial, or may affect the coronary arteries in particular. But, like other affections associated with angina, chronic myocarditis is of frequent occurrence without any manifestation of anginal symptoms.

In some cases sclerotic degeneration is situated outside the coronary arteries, yet in such close proximity to their mouths that later they become affected by contiguity. Thus, chronic aortitis with the various forms of coronary disease and dilatation of the aorta act as causal factors.

Adherent pericardium is sometimes a cause when associated with disease of the aortic valves. Aneurysm of the aorta, either sacculated or fusiform, may also give rise to paroxysms of true angina.

Syphilis in the tertiary stage is an ætiological element of importance. When syphilis attacks the heart it is prone to locate in the aorta and walls of the coronary arteries and induce arterio-sclerosis and thus indirectly cause angina pectoris.

Acute infections and fevers may be attended with angina pectoris, especially in the instance of influenza, where it has been known to follow as a serious sequela. Most of the cases of this type, however, may be more properly classified with the pseudo variety.

The exciting causes are of great importance. They are, muscular exertion, mental emotion and digestive disturbances. These factors act sometimes in a mechanical way. A dilated stomach or intestines distended with gas may press upon the heart and induce an attack. A certain position unconsciously assumed may have the same effect. Physical exertion, especially after a meal, mental emotion, whether grief or joy, anger or any form of excitement may likewise form decided exciting causes.

PSEUDO-ANGINA.—This depends upon direct, reflex, neurotic or toxic influences. It is decidedly more frequent in women than men, and those affected are younger than in true angina.

Direct causes are those which act upon the cardiac nerves by pressure. They are aneurysm, enlarged glands, affections of the brain and spinal cord which implicate the origin of the nerves supplying the heart and neoplasms of the neck and chest.

Reflex causes are those which arise from morbid conditions in other organs. Gaseous dilatation of the stomach or colon may press upon the heart and in nervous persons so interfere with its action that extreme pain arises, attended with dyspnœa and even collapse and syncope. Disease of the liver has been known to cause similar conditions, likewise uterine and ovarian disease. Some observers have thought that the origin of the anginal attacks was in the abdominal viscera.

Peripheral irritation may also be a cause. Cases are on record which have arisen from severe cervico-brachial neuralgia of traumatic or spontaneous origin.

Neurotic influences, while including those of a reflex nature, embrace in addition the effects of emotion in neurotic and hysterical persons, the consequences of parturition, lactation, abortion, anæmia, or a lowered state of the system from any cause.

Toxic agents include tobacco, coffee and tea. Tobacco angina is the most frequent of the toxic forms, although in itself it is not of common occurrence. It was first pointed out by Huchard. The heart is very susceptible to the action of nicotine, as is well known, and in the case of angina it produces a condition of neurosis or spasm. Huchard divides tobacco angina into three forms: First, that due to a functional spasm of the coronary arteries, produced by direct action of the drug on the vaso-motor mechanism of the heart. Second, that due to an arterial spasm caused by tobacco dyspepsia. Third, that due to arterio-sclerosis with narrowing of the lumen of the vessels produced by chronic tobacco poisoning. Thus it will be seen that tobacco may be productive of both true as well as pseudo-angina.

Important exciting causes of pseudo-angina are mental emotion, physical exertion, disturbances of the alimentary tract, chilling of the surface, cold immersion, etc.

Of the causes which are productive of pseudo-angina pectoris, those of reflex origin are the most frequently encountered.

Morbid Anatomy.—TRUE ANGINA.—In all the anatomical changes the main features are embarrassment of the coronary circulation and consequent diminution of nutrition. Yet there are no pathological changes which are invariably attended by symptoms of true angina pectoris.

It has been stated that atheroma of the coronary arteries is the chief cause, hence it follows that the pathology of coronary arterio-sclerosis and that of the causal diseases which underlie it belongs essentially to angina pectoris. On the other hand, arterio-sclerosis of the coronaries is by no means always attended by angina, for numerous cases have been found which were entirely free from all symptoms of such a nature. It is this negative side of the pathological processes associated with true angina which lends obscurity to the question. Jenner, the discoverer of vaccination, first pointed out that occlusion of the coronary arteries was a probable cause of angina pectoris. This theory is now warmly advocated. Numerous autopsies of those who have died of angina show partial or complete occlusion of the coronary arteries, or of its branches, from narrowing due to the presence of calcareous discs or from thrombi or emboli.

In a large number of fatal cases, which have been examined after death, fatty degeneration has been found with disease of the coronary arteries. Frequently the arteries were in advanced stages of calcification and the fatty changes excessive. On the other hand, the changes may be very slight, appearing as yellow striæ in the walls of the ventricles and papillary muscles. Microscopical examination, however, may reveal the presence of fat globules.

The association of chronic interstitial myocarditis in the ætiology in angina points to the fact that the morbid processes which occur with this condition must also be regarded as elements in the pathology of angina pectoris.

While recognizing the close connection between diseases of the coronary arteries and myocardium and true angina, the important and very apparent fact must not be lost sight of, that only a comparatively few coronary diseases are attended with angina, that these affections are common and angina is not. No sufficient reason has been discovered for this variability.

The seat of the pain is doubtless in the heart, but the condition of that organ during the paroxysm and the cause of the pain are more or less subjects of speculation.

Heberden maintained that the heart was in a condition of spasm. Spasmodic contraction of hollow and tubular organs, as in the instance of the bile duct, ureters, intestines and uterus in parturition, furnish striking analogies to bear out this idea, which has found many adherents. On the other hand, it is urged that the true condition of the heart is exactly the opposite, namely, that of paralysis, that this arises from disturbance of the heart rhythm from contraction of the areas surrounding anæmic infarcts, while the affected areas themselves remain incapable of contraction. In cases characterized by a repetition of paroxysms during a brief interval of time, it is not to be supposed that there is a repetition of the infarctions, but that the presence of an infarct in the excited state of the heart causes a diminution of the blood-supply. It is apparent how such a condition causes weakness of the heart and syncope.

Distention and stretching of the heart walls is, according to Traube, accountable for the pain. This observer held that the nerve filaments on the surface of the heart are thrown into a state of tension. The blood-vessels which are able to sustain the heart, when acting quietly, are unable to meet additional demands for more blood when the organ's increased action calls for it. The result is that certain muscular fibres fall into temporary disuse. The consequence is that the increased intracardiac pressure stretches the weakened muscles and at the same time the nerve fibres, hence the pain. The subsidence of anginal attacks in the presence of dilatation, to which attention has been called by Musser, goes to bear out the theory. Musser further states that probably it is on account of dilatation that angina does not occur with mitral insufficiency and that its infrequency among the young and in females is owing to the greater elasticity of the heart walls permitting dilatation.*

Again, it has been contended that the cause of the pain lies in the vessels themselves and is due to angeiospasm or thrombus of the coronary arteries. This theory was set forth in the early part of the last century by Allan Brown.† It is a recognized fact that ischæmia may give rise to the most violent pains, as in the instance of gangrene from embolism and in the treatment of aneurysm by compression, especially at the time of occlusion. It is argued thus—that occlusion of the coronary vessels induces angeiospasm and that the attacks of pain may be repeatedly occasioned by the presence of a single point of obstruction.

The theory that neuritis is the cause of the pain has many supporters. This view in various forms also dates from the early part of the last century. Laennec held that either the pneumogastric or sympathetic division of the cardiac nerves was probably involved, together with the brachial plexus. Against this theory is the fact that aneurysm, mediastinal neoplasms, adherent pericardium and exudative pericarditis may seriously implicate the cardiac nerves without causing pain.

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* Angina Pectoris in its Relation to Dilatation of the Heart, H. Musser, M.D., American Journal of Medical Sciences, vol. cxiv., 1897.

† Quoted by Osler, Angina Pectoris and Allied Affections, New York, 1897.

The state of the arterial system and the blood-pressure have been the subject of considerable discussion. Some observers hold the opinion that there is a condition of angeiospasm and that the blood-pressure is increased. It should be remembered that the exciting causes are such that tend to raise the pressure of the blood. Notwithstanding this, certain cases show a contrary condition. According to Lauder Brunton the pressure is high. Broadbent regards it as variable.

Functional or pseudo-angina has no morbid anatomy.

Symptoms.—TRUE ANGINA PECTORIS.—In typical cases the symptoms are decidedly characteristic. Without warning, the patient is seized with pain of great intensity in the præcordial or sternal region. This pain radiates down the left arm, sometimes the right, as well as across the shoulders and up the back of the neck to the top of the head. In rare instances it may involve the whole chest or may radiate to the epigastric region, abdomen and even the testicles. Sometimes it does not radiate, but is confined exclusively to the arms. The pain is described as of constrictive nature, as if the heart were grasped in a vice or as if it were being crushed against the spinal column; there is a sense of suffocation and inability to move. A characteristic feature is the terrible mental anguish, *angor animi*, the sense and apprehension of impending death.

When the attacks come on the patient suddenly stops whatever he is doing. If walking or standing he seizes the nearest object for support and remains motionless. The attacks make their appearance more frequently in the daytime rather than in the night. They are seldom periodic.

During the attack the patient instinctively seeks the posture which affords the most relief. Sometimes this is sitting, sometimes standing, sometimes sitting and bending over toward the affected side with the hand pressed against the chest walls. The countenance is pale, pinched and staring (*facies Hippocratica*), forehead or entire surface cold or bathed in perspiration. As a general rule, he is speechless or says but little. Sometimes there is vomiting.

Vaso-motor disturbances generally attend the paroxysms of true angina. The arms and hands may become cold, pale, bloodless (local syncope) and covered with perspiration, with often a sensation of tingling or swelling. Cases have been reported in which the pallor has been followed by cyanotic patches in the arms and hands.

The duration of the attacks is from a few seconds to several hours, the average being from five to ten minutes. Recovery is not infrequently attended with eructations of gas and the voidance of large quantities of pale urine of low specific gravity.

The paroxysms may be repeated or the first one may result in death. The patient is always in great peril. More commonly there are recurrent attacks during a period covering several months or even years, in which the termination is almost always sudden death.

After the attack the patient may appear in his former health or he may suffer from cardiac asthma and a tendency to or actual recurrence of the anginal attacks with general enfeeblement of the heart action.

True angina presents decided variations in intensity. In some instances the paroxysms are comparatively mild. There are also variations in type, of which the following are the principal examples :

Angina sine dolore is that form in which the prominent symptoms of true angina, the constrictive oppression, the angor animi and vaso-motor disturbances are present, but the pain is absent. In this connection it is well to recall again the observation of Musser in regard to the relation of dilatation to angina, namely, that as dilatation develops in persons who are subject to angina, the pains grow less and cease. In such instances the change results in the condition known as angina sine dolore, and is likely to be followed by sudden death.

Another type of cases is that where the patient suddenly presses his hand to his side, makes some exclamation referring to the terrible pain in the heart and suddenly expires.

Osler speaks of two anomalous varieties which he calls syncope anginosa and Adams-Stokes syndrome. The former consists of paroxysmal attacks which resemble angina sine dolore, in that faintness is the predominating symptom. The patient does not, however, entirely lose consciousness. In this connection Broadbent is quoted as follows: "A patient who has ceased to suffer with attacks of angina may have attacks of what he calls faintness, in one of which he ultimately dies."*

The Stokes-Adams syndrome is described as follows: "Permanent slowness of the heart action, vertigo and syncope are the two distinguishing features. The cerebral symptoms are naturally those to which chief attention has been drawn. Vertigo is the most common and is usually transient and repeated. Actual syncope of three or four minutes' duration, resembling closely syncope anginosa, and doubtless of the same nature, has been the special feature of some cases, while in others the attack has been apoplectiform in character. Huchard regards the slow pulse as the result of change in the vagi centres due to disease of the arteries of the medulla. The temporary circulatory disturbance of the brain accounts for the syncope and apoplectiform attacks, conditions not rare among aged persons with arterio-sclerosis."†

Still another type of angina is that sometimes observed in aged people with arterio-sclerosis. This is characterized by pain of more or less intensity in both arms and across the shoulders, with some præcordial distress and great general prostration. The mental symptoms and attitude characteristic of true angina are lacking. The pulse is irregular and feeble. Attacks of this nature have been observed by the writer as an accompaniment of the general break-up of old age and have proved to be a precursor of dissolution.

PSEUDO- OR FUNCTIONAL ANGINA PECTORIS.—In this variety the manifestation occurs in several forms and in different degrees of intensity and gravity. The term pseudo, according to Walshe, covers those types of angina which occur independently of any recognizable lesion, but with our still limited knowledge of the subject it is not altogether appropriate.

Anginal paroxysms of this nature are not, as a rule, traceable to exciting causes, but occur spontaneously. The pain is less severe and more diffused over the præcordial region. It is sometimes described as a sensation of distention. The patient is restless and emotional.

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* Diseases of the Heart Considered from the Standpoint of Diagnosis, W. H. Broadbent, M.D., British Medical Journal, 1891, p. 747.

† Angina Pectoris and Allied States, Wm. Osler, N. Y., 1897.

There are four principal forms of pseudo-angina, namely, the reflex, the hysterical or neurasthenic, the vaso-motor and the toxic.

Reflex angina pectoris was first observed by Langlois. It is associated with visceral or peripheral disturbances. The most characteristic cases have been those in which there were decided indications of gastro-intestinal irritation from indigestion. The attacks may be painful or syncope may be the more pronounced symptom. Severe peripheral neuralgias have been known to act as exciting causes.

Hysterical angina pectoris is of a protean type. In conjunction with the anginal paroxsyms some of the characteristics of hysteria are present. There may be severe radiating pain in the præcordial region and vaso-motor disturbances. There may also be hyperæsthesic areas and pains in other areas which alternate with that in the præcordium, also decided evidence of neurotic disturbances elsewhere.

Vaso-motor angina was described by Nothnagel. It is an unusual form. It is characterized by sudden coldness and pallor of the extremities which is frequently attended with profuse perspiration, severe præcordial pain and distress, with palpitation and faintness. It will be observed that these symptoms occur in cases associated with structural lesions of the heart, but they also arise apart from any recognized lesion. In the latter instances they have been designated as vaso-motor angina pectoris. Nothnagel attributed vaso-motor angina to constriction of the arterial system, which acted directly upon the heart. A distinguishing feature of the form is the fact that it is excited by the application of cold to the surface, even washing the hands in cold water. It is characterized also by its proneness to appear in persons of a neurotic and hysterical temperament.

Toxic angina pectoris is limited to that produced by tobacco. It is designated by some writers as a form of true angina, but that is only the case when tobacco poisoning has induced arterio-sclerosis. Huchard, who has written very fully on this subject, mentions a *sclero-tabacique* and a *spasmo-tabacique*. Both of which are rare.

The functional (spasmo-tabacique) which is the most common of the two, is due to spasmodic contraction of the coronary arteries. Balfour* denies that tobacco angina arises from the special action of the poison, but considers it the result of ischæmia from the lowered vitality and impoverishment of the blood from the influence of tobacco. When one takes into consideration the susceptibility of the heart to nicotine, the direct effect of the poison seems the most plausible theory for explaining the cause of the disturbance.

The principal symptoms of tobacco angina, according to Huchard, are vertigo, tendency to syncope, pallor, contracted pulse, coldness of extremities and perspiration, with præcordial distress, which may or may not be attended with pain. These, it will be observed, are symptoms of the vaso-motor variety. Other symptoms pointing to tobacco poisoning, either in association with the paroxysm or independent of them, may appear, namely, gastric disturbances, oppression, dyspnœa, general prostration, mental confusion, disorders of vision, ringing in the ears and spinal hyperæsthesia.

Those who are subjects to tobacco angina almost always manifest during the course of the paroxysm, or apart from it, disturbances of the heart action, namely, feebleness of the pulse, slowness, and acceleration and intermittency, palpitation, tendency to syncope and irregularity of the heart (delirium cordis).

The paroxysms are frequently very severe and complete in their manifestations. But it is in this form of angina that the incomplete or abortive types are observed. These consist of dyspnœa with slight præcordial distress with the sensation of cessation of the heart and apprehension of death. Tobacco angina is further characterized by the spontaneity of the paroxysms; at the same time they may be induced by any motion or effort.†

PHYSICAL SIGNS.—There are no distinctive physical signs attending angina, neither is there necessarily any in the heart-sounds, unless organic disease is present.

Diagnosis.—The characteristic features of true angina pectoris are the sudden, intense pain, substernal and radiating, the mental anguish, and sensation of impending death. The positive recognition of arterio-sclerosis, myocardial degeneration and aortic disease constitutes a most reliable guide to diagnosis. Features of secondary importance are the abrupt cessation of the paroxysm and its excitation by movement of the body.

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* Clinical Lectures on Diseases of the Heart and Aorta, G. W. Balfour, M.D., London, 1876.

† Traité Clinique des Maladies du Cœur et des Vesseaux, Henri Huchard, Paris, 1893.

It is of paramount importance to distinguish between angina dependent upon organic disease and that of functional or neurotic origin. In many instances this is not difficult, but in others it is sometimes the reverse.

The history, age, sex and the nature of the paroxysms must all be taken into consideration. Unless associated with aortic disease, aortitis, or aneurysm, true angina is seldom seen before forty-five years of age. Sex is also an element in the diagnosis, as the affection is extremely rare among women except in the instances just mentioned, while the neurotic type is comparatively common in females over thirty. The presence of a lowered condition of the system in women from any of the causes discussed in the ætiology points strongly to the pseudo variety.

In the hysterical forms the pain is usually diffuse, involving, in some instances, the whole left side of the body in contradistinction to the more confined area of true angina. The discovery of hyperæsthesic areas, hystero-genous regions, clavus, etc., and the presence of palpitation will establish the neurotic element.

Moreover, the patient in hysterical angina is emotional and inclined to walk up and down the room emitting exclamations of distress. The direct reverse of the immobility of angina of organic origin.

In cases of a purely vaso-motor type the history of excitation by cold, the prominence of the vaso-motor symptoms, coldness and tingling of the extremities, numbness, especially of the fingers (local syncope), should direct attention to spasm of the arteries as the cause of the phenomena.

In tobacco angina, the history of excessive use of tobacco with the evidence of chronic nicotine poisoning, as manifested by the characteristic cardiac symptoms, gastric and nervous disturbances, points to the true condition.

Neurasthenic types appear in younger persons, and are recognized by the presence of phenomena characteristic of that condition, namely, paræsthesia, hyperæsthesia, agoraphobia, etc.

In the reflex forms in addition to the neurotic symptoms there is very often some functional disturbance of the alimentary canal, as shown by indigestion, dilatation of the stomach and intestines, vomiting and flatulence. The possibility that the reflex conditions may be of pelvic origin is not to be forgotten. Intercostal neuralgia and gastralgia may to a certain extent simulate angina, but it is scarcely possible that either could be mistaken for it. Attacks of cardiac asthma in association with pain in the præcordial region are more likely sources of error. In true angina, however, dyspnœa is not a prominent nor an essential symptom, while in cardiac asthma the pain, if present, is secondary to the difficulty in breathing. Moreover, in the latter condition, there will be evidence of venous stasis.

The following is a summary of the most important points in the differential diagnosis:

TRUE ANGINA PECTORIS.	PSEUDO-ANGINA PECTORIS.
Ætiology frequently recognized in the presence of arterio-sclerosis and aortic disease.	Ætiology often traceable to neurasthenia, hysteria, tobacco and reflex irritation.
Occurs generally after forty to forty-five years of age.	May occur at any time of life.
More frequent among men.	More frequent among women.
Rarely periodic.	Often periodic.
Induced by effort or exertion.	Generally instantaneous in onset.
Pain intense, constrictive, substernal and usually localized at the level of the aorta.	Pain generally distensive and more diffuse over the cardiac region.
Patient motionless and silent.	Patient agitated, restless and emotional.
	Neurasthenic and hysterical symptoms present.
Prognosis unfavorable, generally fatal.	Prognosis favorable, generally recovery.

Prognosis.—During the paroxysms of true angina pectoris the patient is always in great peril, and it is impossible to foretell the outcome. Some die in the first attack, others may live through a number until one appears which terminates in death.

Angina dependent upon arterio-sclerosis and chronic interstitial myocarditis is most grave. The association of aortic insufficiency is an additional element of danger, but the presence of other forms of valvular lesions is not in itself necessarily of serious significance. Many of the most serious forms of angina are free from any evidence of valvular disease. The existence of chronic nephritis is unfavorable. Recurrence at short intervals of severe attacks, especially if induced by slight exertion, must be regarded as of serious import.

Notwithstanding its gravity, recovery is possible and there are instances in which the attacks have been entirely everted.

In the varieties which fall under the head of pseudo-angina the prognosis is seldom grave and on the removal of the exciting cause the attacks generally disappear.

Treatment.—The treatment of angina divides itself into that of the paroxysms and that of the intervals.

TREATMENT OF THE PAROXYSMS.—*Amyl nitrite* is of first importance. From three to five drops, according to the severity of the attack, should be placed on a handkerchief and the patient allowed to inhale the fumes. The remedy is especially indicated where there is increased vascular tension, but has been extensively used in all forms. Inhalation produces a sense of fullness in the head and a feeling of intoxication without loss of consciousness. It should be discontinued as soon as relief is obtained, or when the pulse and respiration have much accelerated.

Nitroglycerin, although given more as a preventive, is also administered during paroxysms. Compared with amyl nitrite, it is much more lasting in its effects, but its action is much slower. On this account it is best given hypodermically. The one per cent. alcoholic solution, or the second decimal dilution, may be thus administered in one minim doses. The remedy is better suited to milder cases and those of the vaso-motor type.

Morphine is valuable, especially when the pain persists. It may be given hypodermically, in doses of ¼ grain, to be repeated cautiously in half an hour. Some prefer the combination with atropine. Morphine sometimes acts when amyl nitrite fails, or it may prove helpful in association with it. It has the additional property of steadying the heart action, and is especially indicated when the pain is not only persistent, but very severe, the pulse high and respiration accelerated. While caution is necessary in its administration, great tolerance of the drug is sometimes observed.

When amyl nitrite fails, chloroform or ether may be administered. Ether is generally preferred in this country. A few teaspoonfuls in a saucer, and the patient allowed to inhale the fumes, is a suitable method of administration (Romberg). Chloroform is preferred by the British profession. A safe way to administer it is to pour some on a sponge inside a wide-mouthed bottle, place the bottle in the patient's hands, and allow him to inhale; as soon as the effect is produced the bottle falls from the hands (Balfour).

Local applications over the region of the heart are of great service, and should never be omitted. Ice-bags should be tried first, as they are often effective. A recent case under the writer's observation in the Metropolitan Hospital, after the failure of morphine and chloroform, was promptly relieved by this measure. Swallowing small pieces of ice is often useful. Hot applications, on the other hand, suit some cases better.

During the paroxysms the heart must be carefully watched, and any tendency to syncope met with stimulants, such as brandy or whiskey, hypodermic injections of ether or camphor. In some cases digitalis, hypodermically, acts well. Caffeine is preferred by some observers.

When vaso-motor symptoms predominate, and in the forms so denominated, the nitrites are especially indicated and are assisted by hot foot-baths, followed by friction to the extremities. Cold should not be used in these varieties.

In tobacco anginal paroxysms the same general line of treatment is indicated, modified according to the individual demands of the case.

In the neurotic and hysterical angina, the nitrites, and even morphine, may be called for in the severe forms, while the milder attacks may be controlled by ignatia, Hoffman's anodyne, spirits of camphor, valerian, chlorodyne, aromatic spirits of ammonia, and hot, stimulating drinks, such as whiskey and peppermint water, especially if there is much flatulence.

The following remedies will prove useful in those forms of angina where the symptoms do not demand immediate palliation, that is, where the pain is not so intense and the heart well sustained, and in those which have been termed pseudo.

Spigelia, when the pain in the præcordium is of a lancinating character.

Cactus grandiflorus may be administered when the pain corresponds to the characteristic cardiac indication of the remedy, namely, vice-like grasp of the heart.

Veratrum album is advocated in vaso-motor symptoms, coldness of extremities, and perspiration with intermittent, slow pulse.

Lilium tigrum is also indicated for the constrictive pain. Its sphere of action in the female pelvic organs would seem to point to its utility in those forms of reflex angina depending upon irritation of these organs.

Arsenicum is indicated in forms where the patient can only breathe very gently with the chest bent forward, where the least motion causes complete loss of breath, and when stitches and pains in the præcordial region are associated with symptoms of great mental distress.

Nux vomica is useful for reflex intestinal forms, especially in gouty persons.

Among other remedies are aconite in plethoric persons, aconitine suggested by Hughes and mentioned by Goodno, kalmia, cuprum, phytolacca, cimicifuga, rhus, valerian, hydrocyanic acid, naja, spongia, lobelia, tabacum, lactuca virosa and oxalic acid.

After subsidence of the paroxysm the patient may, beyond some exhaustion, appear in his former health, or he may, on the other hand, suffer from decided indications of impaired action of the heart. In some instances acute dilatation results. Even under the most favorable circumstances the patient should remain in bed for several days to a week. In other cases, where the after-effects are prominent, cardiac remedies will be called for, such as nitroglycerin, digitalis, cactus, spigelia and stimulants.

When the paroxysms are easily excited by movement, absolute quiet and rest should be rigidly enforced. Great care should be taken with the diet. The bowels should be kept free.

TREATMENT DURING INTERVALS.—Treatment during the intervals and after the subsidence of the more immediate consequences of an attack includes those measures which are directed toward the exciting causes, which may be termed preventive, and those which are aimed at the primary ætiological factors.

The history of the patient should receive close scrutiny, and any diathetic tendency carefully noted, such as the presence of gout, syphilis, diabetes, or renal disease. True angina in a person under forty years of age should always excite suspicion of syphilis.

Preventive measures embrace the avoidance of gastro-intestinal disorders, especially flatulency, emotional influence and undue muscular exertion. At the slightest intimation of gastro-intestinal disturbance the bowels should be relieved by some saline, calomel or vegetable purgative and the tendency to the formation of gas at once combated.

The questions of diet, muscular exertion and mental strain are those which make up the daily routine of life. How, then, should a person liable to angina live?

Diet is of the utmost importance. Subjects of true angina are very often persons of full habit and hearty appetite which they are inclined to indulge. The amount of food should be cut down and its variety limited, excluding rich and highly seasoned articles and those which favor fermentation. Thus, carbohydrates should be avoided. The dietary usually prescribed for persons of gouty diathesis will in most instances prove suitable. Stimulants should not be taken except in the case of elderly persons who have long been accustomed to their use, then whiskey may be taken with dinner or on retiring.

Exercise must be allowed within certain limitations. Attacks induced by slight muscular exertion, on the other hand, call for a period of complete and absolute rest in bed. In other cases the patient may be allowed to walk on the level, but should avoid fatigue. Walking against the wind should be absolutely forbidden, as well as hurrying and rapid walking. All mental strain, worry and anxiety, close application to business should be avoided as much as possible. Likewise any influence which tends to suddenly affect the emotions.

Medical treatment forms an important part of the measures for the prevention of recurrence of the paroxysm. Not that it can remove structural changes in the heart, but its influence tends to check their further progress.

The most important remedies are the iodides, of which the iodide of potassium is the most favored. It should be given with a copious draught of water after meals in doses of five to ten grains three times a day. Iodide of sodium and iodide of strontium may be substituted for the potassium. Hydriodic acid in the form of the syrup is another effective way in which to administer iodin, the dose is a drachm which contains one grain of the acid. Iodide of arsenic is a cardinal remedy. The tablet triturates in the second decimal trituration, four times a day, is a suitable form of administration. The tendency of the iodides is not only to check the arterio-sclerosis, but also to lower vascular tension.

Arsenic in the form of the triturate, arsenic album or Fowler's solution are also most valuable.

Nitroglycerin is indicated in conditions where the pulse shows a state of high arterial tension. It may be administered for some time, but tolerance is soon established and the dose must be increased. It often acts well immediately after the attack has passed.

The other nitrites are sometimes employed, namely, sodium nitrite and nitrous ether; the former is given in from one to two-grain doses, but is not free from danger; the latter is suitable only for the pseudo forms, especially those of neurotic character.

Aurum in the form of the triturate of the metal or the chloride of gold and soda in the second decimal dilution, five or ten drops three times a day, often proves valuable in retarding sclerotic degenerations of all forms. While especially indicated in arterio-sclerosis of syphilitic origin and in interstitial myocardial changes with symptoms of mental depression, it may be also prescribed from a purely pathological standpoint irrespective of the special indications.

In those types of angina of functional or neurotic origin, the causes should be diligently investigated and their removal sought. It is obvious that no special line of treatment can be laid down, as each will present individual peculiarities which do not allow of generalization.

In tobacco angina it is scarcely necessary to say that the use of tobacco should be prohibited.

In the neurasthenic and hysterical forms little medicine should be given and anodynes withheld. Ignatia, the bromides for a limited period only, the hypophosphites, electricity, massage, the rest cure in a sanitarium and building up of the general health should be the line of treatment followed.

In all instances in both forms of angina, the consideration of the general health is of the utmost importance. Anæmia, general debility and prostration should be met with iron, arsenic, the preparations of cinchona or quinine and strychnine.

ARRHYTHMIA.

SYNONYM.—*Irregular Pulse.*

Arrhythmia is a term applied to disturbed conditions of the heart-beat or pulse. It is needless to say it is a symptom, but its true significance is often difficult to determine. Simple irregularity may exist in some persons, apparently independent of pathological changes, especially those of neurotic temperament. It is also common in children in whom it is more apparent when sleeping. Irregularities of this nature disappear during fever and excitement, while those dependent upon organic change do not. Arrhythmia appears in irregularities of volume and time of pulsation. Its several varieties are as follows:

(*a*) Pulsus alterans; a condition of irregularity in volume and force in which the intervals between the beats are equal, but their volume is unequal; that is, a full pulsation regularly alternates with a weak one. This arises from the contraction of the heart being too feeble to produce a perceptible pulse-wave each time of its occurrence. Pulsus alterans is also generally bigeminus. In some instances the weakened contractions may be so feeble that they produce no pulsation at all and the pulse may appear even.

(*b*) Pulsus intermittans, or intermittance; when one beat is occasionally omitted or is feeble at uncertain intervals. It is caused by weakness of the systolic contractions.

(*c*) Allorrhythmia, a term which signifies variations in the intervals which occur regularly. It appears as the pulsus bigeminus; that is, a regular succession of two beats and a pause, as the pulsus trigeminus, a regular succession of three beats and a pause. Pulsus quadrigeminus may also be observed. The pulsus bigeminus and trigeminus are most frequently found in association with mitral disease. They may also be caused by digitalis; at the same time when originally present they may disappear under its administration. In these conditions the second systole of the ventricles may sometimes be so feeble that the pulse-wave is unable to reach the peripheral circulation and the two systoles are represented by one pulse. The other types of arrhythmical anomalies arise from discrepancies of time, namely :

(*d*) Pulsus paradoxus, that variety distinguished by the peculiarity of becoming weaker or of omitting one or two beats during deep respiration, or toward the end of inspiration. It is an important sign of adhesive pericarditis. It also occurs normally from the effect of respiration on the heart and is sometimes observed in sleeping children.

(*e*) Embryocardia is shortening of the pause between the first and second sounds with a marked similarity of the two, causing the heart-sounds to appear like those of the fœtus. This condition is mostly confined to dilatation and is seen in extreme cases, or in the late stages of long fevers. It indicates excessive weakness of the heart muscle and low arterial tension.

(*f*) Cantering or galloping rhythm occurs when the sounds of the heart resemble the footfalls of a cantering horse, that is, there is a third sound due to reduplication of the second. This may occur at any period of diastole. It indicates weakness and is seen in hypertrophy of arterio-sclerosis, severe forms of anæmia, Bright's disease and acute infectious fevers.

(*g*) Delirium cordis is an excessive degree of irregularity and inequality of the pulse-beats. It is not infrequently observed in valvular disease with dilatation, especially in the late period, and in exophthalmic goitre.

Ætiology.—According to Baumgarten* the causes of arrhythmia may be grouped as follows :

(1) Affections which involve the cardiac centres in the medulla oblongata, viz., central irritation or depression, concussion, cerebral anæmia, psychical influences, etc.

(2) Reflex influences which may originate in some distant organ, notably gastro-enteric disorders, lesion of the liver and kidneys.

(3) Toxic agents, such as tobacco, tea, coffee; certain drugs, such as digitalis, aconite, belladonna, calabar, etc.

(4) Changes in the heart structure as evidenced by the absence

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* Disturbances of the Heart Rhythm with Reference to their Cause and their Value in Diagnosis, D. Baumgarten, M.D., Transactions of the Association of American Physicians, vol. iii., 1888.

of motor power of the organ to perform the work demanded of it. There are many conditions which come under this head; they include the mild grades of incompetency which arise from faulty nutrition of the myocardium, as observed after long-continued fevers and in dilatation of lesser grades, conditions of chronic myocarditis and myocardial degenerations which cover a very wide range of influences, including the more severe lesions, especially atheroma of the coronary arteries and the results of fatty degeneration.

Diagnosis.—The recognition of an arrhythmic pulse at the wrist should be at once followed by a careful physical examination of the heart. Under these circumstances the sphygmograph may prove of special service in determining the variety of arrhythmia. It is obviously very important to differentiate between arrhythmia of functional origin and that dependent upon organic disease. This can be accomplished by carefully examining the history of the case, excluding the various ætiological influences that give rise to the functional forms and by a careful study of the physical signs and symptoms.

Prognosis.—The prognosis of arrhythmia is essentially that of the causes, and is, in consequence, as diversified. Occasionally intermittence of the pulse, as stated, is not infrequently found entirely compatible with health and the patient suffers no inconvenience. Arising in connection with a morbid psychical or functional cause, the outlook is favorable, but in cases dependent upon organic disease it is otherwise. Irregularities of the pulse in diseases of the myocardium or in acute infectious diseases is of serious significance.

Treatment.—The treatment of arrhythmia is that of the many functional disturbances and morbid conditions which enter into its ætiology. Those cases depending upon a lowered tone of the general health and anæmia should receive highly nutritious diet and general tonics. In cases associated with the toxic effects of coffee, tea or alcohol, these agents must be withdrawn and a carefully regulated diet substituted. In such arsenic, nux vomica, ignatia, china, coca and strychnine prove useful. When arrhythmia appears as a symptom of dilatation, digitalis and remedies of its class are indicated. When of neurotic origin or arising from reflex irritation, the remedies suggested for palpitation should be employed.

PALPITATION.

Palpitation is a fluttering, throbbing or violent beating of the heart of which the patient is keenly conscious. It is accompanied by more or less rapidity of action, increased force of the systolic contractions, disturbances of rhythm and præcordial distress and anxiety.

Its prominence as a symptom and the wide range of conditions with which it is associated, varying from slight functional disturbance to grave organic lesion, renders its separate consideration of importance. Mere violent beating of the heart does not always constitute pathological palpitation. Increased action up to a certain limit under violent exercise is physiological; it is only abnormal when it occurs without effort on the part of the patient and when excessive and prolonged. It is more frequently observed in nervous persons.

Ætiology.—The immediate cause of palpitation is overstimulation or excitability of the muscular structure of the heart, from excitement of the accelerator nerve or suppression of the action of the inhibitory nerve. The mechanism of the action is difficult to explain.

The primary causes are direct and reflex nerve irrritation, physical and psychical, altered conditions of the blood and organic disease of the heart. These causes frequently operate conjointly.

Direct irritation includes disease at the origin of the nerves as in organic lesion of the brain and cord and pressure upon the vagus. Thus, palpitation is observed in bulbar paralysis, lateral sclerosis and exophthalmic goitre. Post-mortem examinations have, in several instances, disclosed the presence of ganglionic swellings which had pressed upon the vagus. It is, however, more in its relation to conditions apart from organic disease that the present discussion is directed.

Reflex nerve irritation embraces a variety of disturbances, the most notable being that of digestion, which may be observed in the robust as well as among those of more delicate physique. Mechanical effects may result from the same cause, as pressure of a distended stomach upon the heart. Autointoxication from the absorption of excrementitious matter, as observed in sick headache, is sometimes attended with palpitation. It may also be induced by the ingestion of certain articles of food by individuals possessing idiosyncrasies of digestion, for example, strawberries, shell-fish, etc. Diseases of the female pelvic organs are a common cause, and rectal and urethral irritation in the male are occasionally attended with the same results.

Psychic and neuritic influences comprise emotions of any kind, joy, grief, anger, fear, homesickness, shock, prolonged anxiety, lovesickness, nervous excitement, etc. Persons about to undergo a physical examination, public speakers and actors occasionally suffer from palpitation. The symptom is likewise observed in certain disturbed and exhausted conditions of the nervous-system, as at the menopause and at puberty, in hysteria and as the result of sexual excesses, prolonged continence and unnatural gratification.

Palpitation of the heart frequently appears in connection with altered conditions of the blood, either from impoverishment of nutrition or from the development of toxines which arise in these states. Thus, in the various forms of anæmia and chlorosis it is a common symptom. It also occurs in gout from the presence of the uric acid and the changed condition of the blood-vessels. In Bright's disease, especially in connection with cardiac hypertrophy, it is not infrequently observed. It likewise sometimes occurs in tuberculosis. Acute infections are also in many instances attended with palpitation.

Toxic influences include the excessive use of alcohol, which is one of the most frequent causes, coffee, tea and tobacco. Palpitation is especially common among those who drink excessive quantities of tea, which acts in disturbing the digestion as well as a toxine.

Symptoms.—The principal feature is obviously the intensified and tumultuous action of the heart which is felt by the patient, and is variously described as a fluttering, violent pulsation, "a pumping or turning over" of the organ, or as if it were pushing up into the throat. In pronounced cases the heart beats violently against the chest walls, which show decided agitation. The pulse is often rapid, sometimes running as high as one hundred and fifty per minute. There is usually anxiety, fear and difficulty in breathing. Pain in the præcordium, which is generally relieved by pressure, is not unusual. Nervous symptoms may be present, which vary according to the associated conditions. In attacks due to disorders of the digestion there are eructations of gas and other evidences of gastro-intestinal disturbances.

In severe forms there is decided dyspnœa, difficulty in speech, ringing in the ears, headache, vertigo and great apprehension. The countenance is pallid and bathed in cold perspiration. There may be even partial syncope.

Palpitation of the heart is paroxysmal. The paroxysms continue for a period varying from a few minutes to several hours, with or without intermissions. In exophthalmic goitre it is most persistent. Generally, the paroxysms appear in the daytime, but occasionally they come on at night, when they often prove a source of much anxiety and sleeplessness. Their onset and decline is exceedingly sudden. After an attack there is considerable languor and prostration, both mental and physical. When the paroxysms are of gastric origin there may be excessive hunger.

In cases of a purely neurotic origin, the patient's countenance may show good color and his general appearance presents a favorable contrast to the distress from which he suffers.

Apart from the tumultuous action of the heart, as seen by inspection and observed by palpation, and exaggeration of the heart-sounds, palpitation does not possess characteristic physical signs.

Diagnosis.—The diagnosis is very readily made, the condition being at once apparent. The chief interest is the discovery of the cause and the determination of the presence or absence of organic heart disease. The association of anæmia, nervous affections, dietetic errors, emotional influences, neurasthenia, organic disease of the brain and cord, the abuse of tea, coffee, alcohol, tobacco, etc., must all be taken into consideration. In some instances the cause is very obscure.

Palpitation arising from organic disease of the heart is usually excited by exertion and the cause, it is needless to say, is recognized by the physical signs.

Prognosis.—The prognosis is generally good. The outlook, however, depends upon the recognition of the cause and the ability to remove it. In palpitaiton arising in connection with organic diseases of the brain, cord or valvular system it is less favorable.

Treatment.—During the attack the patient should be placed in a semi-recumbent posture in an apartment with plenty of fresh air. The clothing around the throat and chest should be loosened. An ice coil, or, in its absence, iced cloths may be placed over the præcordium. Stimulants, in the form of brandy, whiskey and aromatic spirits of ammonia should be administered. If the nervous element predominates ignatia, Hoffman's anodyne, moschus, nux moscata, chamomilla, coffee, aconite, spigelia, valerian, valerianate of ammonia and bromide of soda will prove of service. Gelsemium is recommended for tobacco palpitation. In extreme cases morphia, hypodermically, in doses from one-eighth to one-quarter of a grain, is useful. If there is præcordial pain, sinapisms may be applied. Electricity in the form of a constant current is also useful. The positive pole should be applied at the inner border of the sterno-cleido-mastoid muscle and the negative to the lower border of the sternum. A moderate current of a few minutes' duration should be used.

Posture may also prove helpful, viz., the body bent, arms by the sides and breath held for a few seconds (Brunton).

After the subsidence of the paroxysms a careful examination should be made to determine the cause, and treatment should be instituted for its relief and removal.

TACHYCARDIA.

SYNONYMS.—*Recurrent Tachycardia, Rapid Heart, Heart-Hurry, Polycardia, Pyknocardia.*

Tachycardia is a term which was first employed by Gerhardt, in 1881, to designate extreme rapidity of the heart action. It is a symptom complex of obscure origin, generally appearing in paroxysms. Bouveret[*] for this reason described it, in 1889, as recurrent tachycardia. It is sometimes, though not always, unattended with subjective symptoms.

It should be borne in mind that the pulse-rate of some persons is physiologically higher than the usual normal standard and it is claimed that certain individuals can increase the pulse at will. It is said that Sir William Congreve's pulse was 120 per minute.

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[*] Tachycardia Essentielle Paroxystique, L. Bouveret, M.D., Revue de Medicine, Paris, 1889.

Ætiology.—Although much obscurity surrounds the origin of tachycardia, there are three general groups of conditions which are regarded as factors in its manifestation. There are first, neurotic influences, including those which are productive of palpitation, viz., hysteria, neurasthenia, general exhaustion, debility, and reflex irritation; second, anæmia and other altered conditions of the blood; third, nerve irritation, which in turn appears in two varieties, central, as in the instance of cerebral tumors and clots, degenerations of the medulla and cord, and peripheral, where the vagus is subjected to pressure from aneurysms, enlarged glands and tumors, or when it is the subject of neuritis. In the larger proportion of cases sudden exertion appears to be the exciting cause.

Heredity, according to Œttinger,* exerts a predisposing influence. In regard to age and sex, Herringham† found that in fifty-three cases which he collected, thirty were in males and twenty-three in females, and that in the great majority of instances the affection began to manifest itself after twenty years, only seven cases appearing in childhood.

Morbid Anatomy.—According to Gibson, only six cases of tachycardia have been examined after death; of these one showed fatty degeneration of the heart muscle, two, chronic interstitial myocarditis, and three, cardiac dilatation.

The mechanism by which the affection is induced remains up to the present time in obscurity. Many theories have been advanced. Tuchzel ascribed the phenomena as due to paralysis of the vagus; Debove called it a bulbo-spinal neurosis; Talamon suggested epilepsy. The weight of post-mortem evidence, although too limited to allow of the formation of definite conclusions, points to the myocardium as the seat of the lesion with probable implication of the nerve terminals. It is important to note, however, that no evidence of nerve lesions was discovered in the six autopsies above mentioned.

Symptoms.—The essential feature of tachycardia is the extremely rapid pulse-rate which appears suddenly in paroxysms, usually without premonition. Præcordial distress and constriction, tingling and numbness of the arm, palpitation and pain are occasionally present, but are not essential symptoms. There may be vertigo, exaggerated pulsation of the carotids or pulsation of the jugulars. The respiration is usually increased, but only in rare instances is there dyspnœa. The countenance is generally pallid, but sometimes may be cyanosed.

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* De la Tachycardie Essentielle Paroxystique, M. le Docteur Œttinger, La Semaine Medicale, Paris, 1894.

† Concerning Paroxysmal Tachycardia, W. P. Herringham, M.D., Edinburgh Medical Journal, 1897, vol. i.

The main feature is the pulse-rate which runs as high as 160 to 200 or even 300 per minute. The arteries are somewhat empty and the pressure is low, sometimes the pulse is irregular.

The attitude of the patient during an attack is variable. In some cases there may be apparent unconsciousness of the condition, or there may even be a sense of slowing of the heart, in other cases the patient may lie speechless and helpless, the surface cold and with every evidence of intense suffering. There may be some pulmonary hyperæmia, œdema of the subcutaneous tissues and albuminuria.

The attack ceases as abruptly as it comes on. The face resumes its natural color, the surface of the body, its warmth and the pulse falls to normal ; in some instances it may fall considerably below the normal standard. Profuse diuresis and diaphoresis or diarrhœa may accompany a paroxysm or mark its termination.

The duration of the attacks varies from a few minutes to several hours or even several days. Some cases have been reported which have presented very curious features. One of Eccles' cases was relieved by flexing the right knee on the abdomen ; one of Bristowe's by putting a pillow under the loins. H. C. Wood reported the case of a physician eighty-seven years of age who had been subject to attacks since his thirty-seventh year, the pulse running up to 200. The attacks were said to be arrested by drinking ice water or strong coffee.

Physical Signs.—These are in no way distinctive. The area of cardiac pulsation will be found to be increased and the force of the pulse diminished, there may be some increase in the area of dulness and loss of intensity of the sounds. Some writers mention accentuation of the first sound, lessening of the intensity of the aortic second sound and increase of the pulmonic second sound. Adventitious sounds may be present.

Diagnosis.—The presence of a pulse-rate in the neighborhood of 160 and the absence of dyspnœa usually permit the diagnosis to be made without difficulty.

From palpitation, tachycardia is distinguished by the greater rapidity of the pulse and the comparative or entire freedom from præcordial distress.

Prognosis.—The future is uncertain. Some patients suffer no severe derangement of health and may live for years. On the other hand, there is a tendency for the condition to become intensified as the years advance. After thirty years of age, according to Herringham, the patient is never safe.

Cure in most instances is more than doubtful. When the cause can be removed it is sometimes effected, though not always. Death may occur from heart failure and, in the case of those advanced in years, from cerebral hæmorrhage.

Treatment.—The management of tachycardia depends upon the individual peculiarities of each case. If dilatation is present the use of the Nauheim baths often proves of benefit. If there are evidences of degeneration of the heart walls, treatment should be directed toward this condition. Treatment for the paroxysms should be on the same lines as for palpitation.

BRADYCARDIA.

Bradycardia is a condition of excessive slowness of the heart action. It is the antithesis of tachycardia, but resembles it in that it is a symptom complex. Like it, also, the term is applied by some writers exclusively to purely functional disturbances, but, inasmuch as its origin in many instances may be traced to organic disease as well, the question will be discussed in its relation to both functional disturbance and pathological changes.

In determining the frequency of the pulse, that at the wrist should be compared to the actual contractions of the heart. In this connection, it is important to note the observation of Hope,* that in some instances of slow pulse one or two systolic contractions may regularly fail to reach the radial pulse. This is the false intermission of Laennec. Again, in some individuals the pulse is normally far below the usual standard. As is well known, according to Corvisart, Napoleon's pulse was only forty. Roux relates the case of an agriculturist who had passed through his military service without difficulty and who was apparently in excellent health, yet his pulse was only thirty-four to forty per minute. Active exercise, such as running, would not make it ascend higher than fifty-five per minute.

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* Treatise on Diseases of the Heart and Great Vessels, James Hope, M.D., 3d ed., London, 1839.

Bradycardia may be paroxysmal or recurrent, temporary and permanent. The first and second varieties may signify either functional disorder or organic disease, the third, organic disease alone.

Ætiology.—Paroxysmal bradycardia makes its appearance abruptly in persons in apparent health and disappears after a short interval. It is a neurosis dependent upon reflex and toxic influences. It may be seen in gastro-intestinal disturbances, nervous exhaustion, as from mental or nervous strain, or sexual excesses and the abuse of coffee, tea and alcohol.

Temporary bradycardia implies the presence of a slow pulse which persists for a longer or shorter period. It differs from the paroxysmal in that there is a greater degree of persistence. It arises in connection with acute infections, such as rheumatism, typhoid fever, diphtheria, pneumonia, etc., especially in the young with constitutional diseases, such as anæmia and chlorosis, in diabetes, icterus and the puerperal state, and occasionally with nephritis and uræmia. In very rare instances it occurs with emphysema and asthma and affections of the skin and sexual organs. The causal factors which are operative in the paroxysmal variety must also be included under this head, especially gastro-intestinal reflexes, nervous exhaustion from any cause, and the abuse of tobacco, coffee, tea and alcohol.

Permanent bradycardia arises from organic disease of the heart and of the nervous-system. When of cardiac origin it usually implies the presence of degenerative conditions of the myocardium and arterio-slerosis of the coronary arteries, but in only a small proportion of these cases, however, does it appear. In disease of the valves it is seldom observed except in aortic obstruction, when it may occur as the result of mechanical pressure. In arterio-sclerosis of the coronary arteries with occlusion from embolism or thrombus, the pulse suddenly falls very low, in some instances to only eight or ten a minute, and steadily so continues till death. A sudden fall of this nature is usually attributed to this condition, although it may be due to other causes, as, in a case cited by Prentiss, mentioned by Whittaker, in which the pulse fell to five. The patient was a man of fifty-three, whose pulse for one or two years had ranged from eleven to forty per minute. No disease of the heart or other organs was recognizable. Toward the end of his life he became violently delirious and suddenly died. Post-mortem examination showed that the aorta and coronary arteries were free from atheroma, but that the recurrent laryngeal nerve had upon its surface a number of "fusiform enlargements." The cause of the bradycardia was attributed to this condition.

The lesions of the nervous-system where bradycardia appears as a symptom are diseases of the brain and cord, such as cerebrospinal meningitis, meningitis, hydrocephalus, concussion, shock, myelitis, tumors, abscesses, or any condition causing pressure. It is also observed in fractures of the cervical vertebræ, especially of the fifth.

Morbid Anatomy.—It is scarcely necessary to say that bradycardia has no morbid anatomy of its own. Those lesions with which it has been associated have been, in the instances of the heart, mostly of a sclerotic nature. The frequent association of bradycardia with arterio-slerosis has been mentioned. Huchard observes that sclerotic changes in the arteries of the brain, and especially of the medulla oblongata, are not infrequently associated with slow pulse.

Symptoms.—The slow pulse is the essential and determining feature of bradycardia. If paroxysmal, its onset and disappearance are equally sudden. There may be entire absence of any other symptoms and the patient may be unaware that there is anything wrong with the heart action. In a case of the writer, a delicate man of forty-four, after alcoholic and sexual excesses, the pulse fell to thirty-four per minute without the slightest inconvenience to the patient, who was unaware of the condition.

On the other hand, there may be great prostration, vertigo, faintness, difficult respiration and, in severe forms, attacks of syncope with unconsciousness which may last for hours. Some writers mention lapse of memory and convulsive seizures. The dyspnœa may assume the character of Cheyne-Stokes respiration, especially in the paroxysmal form.

The urine is likely to be scanty and may show the presence of albumin and casts. The countenance displays nothing characteristic. It is pale or cyanosed. In some instances the superficial arteries stand out rigid and tortuous. The blood-pressure, as a rule, is low, though in rare instances it may be the reverse.

The pulse-beat varies from forty-eight down to five. The latter was observed by Halberton. It is generally regular, though irregularity may be observed. Præcordial distress is rare.

PHYSICAL SIGNS.—The heart-sounds are characterized by weakness. The first sound may be loud and prolonged, as in hypertrophy, or short and sharp, as in dilatation.

Diagnosis.—A pulse below forty-eight per minute, which corresponds to the systole of the heart, may be regarded as bradycardic.

Prognosis.—The outlook depends entirely upon the cause. When associated with organic lesion of any kind it is of grave significance. In reflex disturbances when the cause can be removed, as in conditions of debility and anæmia, the prognosis is favorable.

Treatment.—In all instances the cause, if possible, should be ascertained. As a symptom, it is evident that a wide range of influences must be taken into consideration. Temporary bradycardia frequently disappears without much interference. Paroxysmal bradycardia requires direct measures for relief. The patient should be kept in a recumbent position and stimulants administered, notably whiskey and ammonia.

In the permanent form, remedies directed to the improvement of the nutrition of the heart constitutes the line of treatment. Glonoine should be considered first. The iodines are also of benefit, especially arsenicum iodide. These remedies should be continued for a very long period. Digitalis does not, as a rule, act beneficially. If the heart shows evidence of sudden failing, strophanthus and strychnine, especially the latter, will be called for as temporary expedients.

CARDIAC NEURASTHENIA, WEAK HEART, IRRITABLE HEART, "SOLDIER'S HEART."

There are a number of affections of the heart which are difficult to define and which, while apparently functional and independent of organic changes, are not always clearly separated from them. They have been described as weak heart, cardiac neurasthenia, cardiac asthenia, irritable heart and "soldier's heart." These conditions are intimately related, all being the results of defective innervation, their differences for the most part being those of type and degree. They have for this reason been grouped by some writers under the general head of neurasthenia cordis, but, inasmuch as they present certain individual characteristics, their separate consideration seems advisable. From their nomenclature absence of any organic change in the structure of the organ is implied. This, however, is not literally true, since modern pathology has demonstrated that in functional disturbances the nerve cells undergo a certain amount of granulation, contraction of the protoplasm and vacuolation of the nuclei. These effects have been observed after nervous irritation and fatigue. They are characterized by their transitory nature.

Cardiac Neurasthenia.—This is a condition of debility of the heart muscle arising from defective innervation of the nervous-system which affects the heart as part of the general process. It occurs usually among the young and those of nervous temperament, but generally of good constitution, and is more frequent in the upper walks of life.

Its ætiology presents a variety of causal influences, notably reflex irritations, exhaustion, both physical and psychical, excesses, onanism, contusions, coitus reservatus, psychical influence, such as shock, grief, failure in business, mental stress of any kind, general neurasthenia, the effects of long fever, especially typhoid, excess of uric acid, gout and suppressed gout. The abuse of alcohol, tea and coffee may act as active contributory causes.

The clinical features of cardiac neurasthenia are usually characterized in the earlier period of the affection by excitement and irritation, and in the later stages by a more or less gradual change to a condition of atony.

The onset of the stage of excitation may be either gradual or sudden, usually it is the former. There is often great increase in the rate of the pulse, which may mount as high as two hundred per minute, being, in fact, a true tachycardia. In rare instances there is bradycardia. The sensation of palpitation is not usual, the patient may rather complain that the heart is acting feebly. The pulse may be intermittent and irregular and easily be affected by change of position or even disturbing thoughts. There is sensitiveness in the præcordial region and intercostal spaces, or there may be præcordial distress, anomalous sensations, anxiety, labored respirations and sometimes severe pains simulating angina.

The countenance is pale or flushed, changing from one condition to the other. It may be red in spots or lines. There is numbness of different parts of the body, especially of the fingers and toes. The extremities may be cold or hot, or both, alternately. The superficial arteries are distended and tortuous. Nervous symptoms and insomnia are more or less pronounced, sleep is disturbed, the patient waking with great anxiety. There is excessive sensitiveness to any extraneous influence, noise or sudden surprise or movement. The heart action may be strong and rapid, but with no signs of organic change.

After a while the symptoms become less active and the period of decline or atonic stage appears. The patient now becomes more irritable or very apathetic or indifferent. The pulse shows lowering of tension of the arterial system, as evidenced by dicrotism. The heart action is further characterized by increase of weakness and irritability. In a certain number of cases, symptoms of general neurasthenia, with a varying degree of prominence, may be present, such as lack of interest in events of life, disinclination to effort and indifference to surroundings, constant introspection, agoraphobia, nosophobia, monophobia, intense irritability, suppressed excitement, neurasthenic pains, etc.

The neurasthenic heart may closely simulate weakness from organic disease, but the absence of the physical signs of organic changes, together with the presence of symptoms above mentioned, will usually remove all source of error. Organic heart disease, however, may cöexist with neurasthenia.

The outlook is generally good, although, as in other forms of neurasthenia, the condition is often prolonged and obstinate. In time, however, the heart and general health may regain their lost tone and restoration be complete. In some cases, on the other hand, especially those in which the course of the affection has been very protracted, the prognosis is not by any means so hopeful as far as complete recovery is concerned.

The management of cases of cardiac neurasthenia is on the same lines as that of general neurasthenia and is, therefore, mostly hygienic and dietetic. Change of scene and climate and diversion or a sea voyage suit some cases. For others the rest cure and isolation is better.

The diet should be highly nutritious and easily digested. The appetite should be stimulated by tonics, such as arsenic, cinchona and nux vomica. When there is an excess of uric acid, alkalies or some suitable mineral water will be of advantage.

For the insomnia, a warm bath followed by a glass of hot milk at bedtime should be tried in preference to the use of hypnotics which, if possible, should be avoided. Among useful remedies are the hypophosphite of lime and soda, the glycerophosphate of lime, kali phosphate in trituration, arsenicum album, ignatia, phosphoric acid, strychnia subcutaneously beginning with small doses and increasing, hydrotherapy with friction and massage, the Nauheim baths and the graduated exercises of Schott.

Weak Heart.—This term is employed in two senses, in the first place it signifies a relaxed and constantly failing organ, in the second a transitory weakness.

The first type of cardiac weakness includes a variety of causes, namely, inadequacy, influences which when more active and severe result in degeneration and atrophy, pyrexia, abuse of alcoholic stimulants, excessive fatigue, congenital weakness, and any condition which may lead to defective nutrition. Post-mortem examination of the heart in this form of cardiac weakness in some instances discloses a condition of simple relaxation of the mural muscular tissues, which are soft and friable, but no change visible with the microscope. In others there is the atrophy of the fibrillæ and augmentation of the longitudinal striæ, with separation of the muscular fibres. Granular atrophy may also be present with decrease of striation, but without diminution of the muscular substance. Pigmentary atrophy of a brownish color may also be observed with pigmentary deposit around the nucleus of the muscle cell. It should be remembered, however, that a certain amount of pigment is present in every heart after death, the deposits being found in that part of the organ which is subjected to the most strain.

The symptoms of this form of heart weakness are shortness of breath, palpitation with some faintness and vertigo. The pulse is small and empty, sometimes very slow and sometimes very rapid, the rhythm is fluctuating, being at times regular, at others irregular. The impulse and heart-sounds are weak and almost, if not entirely, imperceptible. The first sound appears to undergo the greatest amount of diminution in force and in some instances it may not be possible to distinguish it. A soft blowing murmur may be audible in the mitral and tricuspid regions. In severe cases there is œdema and symptoms of venous stasis.

This group of symptoms may be more or less, but not entirely, obscured by those of the morbid condition which gives rise to it.

Another type of this condition arises from nervous strain, such as worry, overwork or any psychical cause. It may occur in either sex. The patient appears entirely prostrated, lies in bed and falls back in a fainting condition at every attempt to sit or stand up. There is more or less insomnia. The heart action is feeble and the pulse small and increased in frequency with disturbance of rhythm.

The future of cases of this type is generally variable and treatment should be on the general principles of removal of the cause and of increase of nutrition.

The second or transitory form of heart weakness is for the most part observed in neurotic persons, especially females. The patient complains of a sensation which is described as if for a moment the heart ceases to beat. The countenance becomes pallid and the hands are usually pressed against the præcordial region in an effort to relieve the throbbing. There is nervousness and apprehension. The pulse is somewhat weak and accelerated, ranging from 100 to 110. The condition is free from danger. Stimulants usually prove effective, but alcohol in any form should be avoided as far as possible for the attacks are recurrent, and the liability of forming the alcoholic habit in these cases is often pronounced.

Treatment should include massage, electricity, salt water baths, strychnia and general tonics.

Irritable Heart.—This condition was first described by DaCosta, being distinguished by him from cardiac weakness, weakness of organic disease, and that of lithæmia, gout and tobacco poisoning. It is observed in the young and also in those of advanced years, but is more frequent in the former. It usually results from the effects of strain, either physical or psychical.

The principal symptom is a sense of throbbing and palpitation. The heart, it is said, appears to suddenly jump. This sensation may be experienced either with or without apparent exciting causes and may even occur during sleep. Although regarded as a separate entity, irritable heart is strongly suggestive of a form of palpitation. There is commonly the history of excess in exercise or some nervous disease. Examination discloses some resemblance to hypertrophy. The first sound is indistinct, the second at the apex somewhat exaggerated. The rhythm may be uneven and the pulse dicrotic. The arteries are lax in contrast to the excited action of the heart.

"**Soldier's Heart.**"—This is an expression which has been applied by certain writers to a condition sometimes observed in new recruits, especially those who have led sedentary lives. It is a form of muscular strain. Allbutt, in his System of Medicine, described the symptoms as follows: "The patients state that while remaining free from any physical exertion they experience no discomfort and feel quite well, but immediately on commencing to march they suffer with throbbing of the heart and shortness of breath, sometimes accompanied by vertigo and faintness. There may also be pain, nervousness or insomnia. Pulse may be either regular or irregular, ranging in frequency from 100 to 120, with low arterial pressure. These symptoms disappear on resting, but reappear on resuming the march."

Physical examination shows no evidences of organic lesion, although, according to Allbutt, there is sometimes increase in the area of dulness with diffusion of the impulse. In two cases it is stated that there was reduplication of the second sound at the base, in three the first sound was prolonged and booming, and in six the first sound was sharp.

SECTION VI.

POSITIONS AND MALFORMATIONS OF THE HEART.

MALPOSITIONS.

n is *dextrocardia*, in which the heart occupies
e chest. In this condition there is frequently
the viscera. The heart may also occupy a posi-
nedian line than normal, a situation peculiar to
is instance it is called *mesocardia*. According to
rt has been found in very rare instances lying in
antero-posterior position.

iety of displacement is that termed *ectopia cordis*, or
acement. In this condition the sternum is deficient
nt and is divided vertically, leaving the heart exposed
integument. There are three forms: *ectopia cordis*, or
ectoralis, in which the organ lies in front of the chest;
bdominalis, in which it lies in the abdominal cavity; and
cephalica, in which it lies at the root of the neck.

MALFORMATIONS.

eart may be entirely absent constituting the condition called
, or it may be too large or too small or defective in parts.

FORMATIONS OF THE SEPTUM.—The septum between the auri-
nd ventricles may be absent, forming two chambers, or *cor*
laris, or it may be lacking between the two ventricles, forming
chambers. More frequently there is a simple perforation
incomplete development which is generally observed in the
terior portion of the interventricular septum. These deformities
frequently accompanied by obstruction of the conus arteriosus
the right ventricle and of the pulmonary artery.

The causes of this condition are given as excessive labor and short rations with excesses in alcohol and tobacco. The prognosis is said to be unfavorable and the condition is liable to result in permanent dilatation. There are no records of post-mortem examination of cases of this nature.

SECTION VI.

CONGENITAL MALPOSITIONS AND MALFORMATIONS OF THE HEART.

MALPOSITIONS.

The simplest form is *dextrocardia*, in which the heart occupies the right side of the chest. In this condition there is frequently transposition of all the viscera. The heart may also occupy a position nearer the median line than normal, a situation peculiar to fœtal life. In this instance it is called *mesocardia*. According to Peacock, the heart has been found in very rare instances lying in a transverse or antero-posterior position.

Another variety of displacement is that termed *ectopia cordis*, or external displacement. In this condition the sternum is deficient in development and is divided vertically, leaving the heart exposed beneath the integument. There are three forms: *ectopia cordis*, or *ectocardia pectoralis*, in which the organ lies in front of the chest; *ectocardia abdominalis*, in which it lies in the abdominal cavity; and *ectocardia cephalica*, in which it lies at the root of the neck.

MALFORMATIONS.

The heart may be entirely absent constituting the condition called *acardia*, or it may be too large or too small or defective in parts.

MALFORMATIONS OF THE SEPTUM.—The septum between the auricles and ventricles may be absent, forming two chambers, or *cor bilocularis*, or it may be lacking between the two ventricles, forming three chambers. More frequently there is a simple perforation from incomplete development which is generally observed in the posterior portion of the interventricular septum. These deformities are frequently accompanied by obstruction of the conus arteriosus of the right ventricle and of the pulmonary artery.

MALFORMATIONS OF THE CHAMBERS.—The chambers of the heart may show congenital diminution or increase in size. The ventricles may undergo dilation and hypertrophy; the latter usually predominates. Hypertrophy of the right ventricle is one of the most frequent anomalies, the walls show increased thickness with decrease in the size of the chambers and the muscular substance distinct proliferation with occasional interstitial changes. The right auricle may be dilated to an excessive degree. Decrease in either of the auricular chambers may also be observed with decrease in the corresponding ventricles.

MALFORMATIONS OF THE ENDOCARDIUM.—The membrane may show the evidences of a fœtal endocarditis. It may be roughened, thickened and studded with vegetations.

A peculiar feature is the presence of nodules attached to the borders of the valves. These are small, fibroid bodies covered with endocardium. Free bodies are also found. They are small, dark and spheroidal in shape and almost the size of a millet seed. They resemble coagula and have been called hæmato-nodules. They are covered with a membrane like the endocardium. These peculiar bodies are found in still-born children and sometimes in others where they have been discovered some days and even years after birth.

Malformations of the chordæ tendineæ also occur. The cords, for example, cross a ventricle and may be attached to a valve. Under these conditions a musical murmur may develop. The condition thus far has only been observed in the left ventricle.

MALFORMATIONS OF THE VALVES.—The segment of the valves may show numerical discrepancies, especially those of the aortic and pulmonary, the most frequent being two segments. Supernumerary segments are usually rudimentary and are more frequently observed at the pulmonary orifice. Deficiencies in the segments, on the other hand, are more common at the aortic orifice. Narrowing of the great vessels is often an accompaniment of these deformities.

Fenestration of the valves may occur. Generally the opening is near the margin. Fusion of the valves is found as the result of fœtal endocarditis. The segments of the pulmonary valve may become agglutinated and project like a funnel into the vessel. The auriculo-ventricular valves may also show anomalies, but these are less frequent.

ANOMALIES OF THE GREAT VESSELS.—The aorta and pulmonary artery may sometimes be found in abnormal situations. They may both communicate with either ventricle or with both, or the aorta may arise from the right ventricle and the pulmonary artery from the left. The trunks of the vessels may also show departure from the normal either in diminution in size or in complete absence.

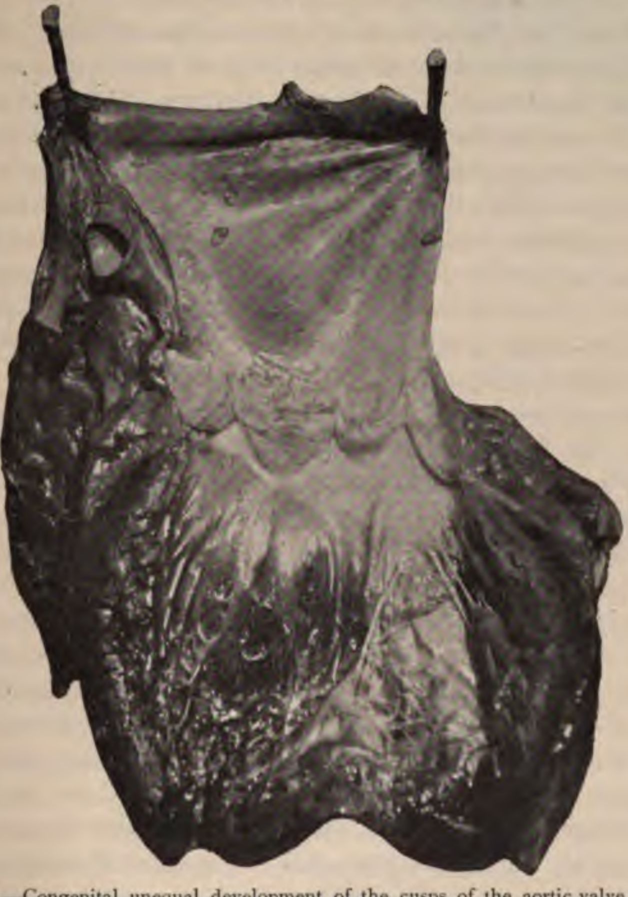

FIG. 35.—Congenital unequal development of the cusps of the aortic valve with no evidence of endocarditis or atheroma of the valve itself. Some atheroma above the valve. The central cusp in the illustration measured 2.5 cm. and each of the others 1 cm. This condition led to insufficiency of the valve followed by hypertrophy and dilatation of the left ventricle.

Defects in the pulmonary artery are among the most common of congenital malformations. Its orifice may be constricted by agglutination of the valve segments, which may form a funnel-shaped body which protrudes into the vessel. In some instances the vessel is entirely obliterated and ends in a cul-de-sac.

Narrowing of the pulmonary artery is also common. It is frequently accompanied by patency of the foramen ovale and sometimes by the absence of the ductus arteriosus. The right ventricle, under these conditions, is always found hypertrophied and dilated. In some cases the whole extent of the pulmonary artery is smaller than normal. When the pulmonary artery is absent or closed and the ductus arteriosus remains, the circulation finds its way from the aorta through the duct into the branches of the pulmonary artery. When the duct is closed the bronchial arteries carry the blood to the lungs.

The aorta may be abnormally small or large, but it does not display defects so frequently as does the pulmonary artery. The orifice may become occluded or entirely obstructed by fusion of the valve. The former is more common.

Smallness or contraction of the aorta is attended with decided hypertrophy of the left ventricle with high tension, which may be followed by dilatation. Murmurs are heard, generally systolic in character.

The ductus arteriosus sometimes remains patent. When one end only is closed the other is found in connection with the pulmonary artery. In cases where the duct remains permanently open the blood finds it way from the aorta to the pulmonary artery, and by the increased pressure thus caused leads to dilatation and hypertrophy of the right ventricle.

Ætiology and Morbid Anatomy.—Congenital anomalies of the heart are the result of arrested or perverted development and of fœtal endocarditis. The manner in which morbid developmental influences affect the growing embryo is shrouded in mystery and, in consequence, is more or less the subject of speculation. Interesting experiments have been made by Geoffroy-Saint Hilaire and Féré. The former, near the middle of the last century, took incubated hen's eggs, shook some violently and coated others with varnish. The results were arrested developments and malformations.*

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* Histoire Generale et Particuliere des Anomalies de l'organization chez Hommes et les Animaux, par M. Isadore Geoffroy-Saint Hilaire, Bruxelles, 1837.

More recently Féré* injected pathogenic germs into incubating eggs and also succeeded in producing malformations. These results point to the conclusion that mechanical interference with the growth of the embryo and disease produce similar effects.

Ballantyne† in commenting upon the results of these experiments, together with those of other observers in the line, such as Dareste and Lombardini, says that substances which act as the cause of disease in extra-uterine life and in late intra-uterine life, act as the cause of monstrosities in early intra-uterine life. The same causes act in both periods, but when the body is performing its functions they lead to disease, and when it is in the process of formation they produce teratological effects. Teratology, he considers, is the pathology of the embryo.

Peacock, who had made extensive research in the line of congenital anomalies of the heart, says that the morbid influences which affect the organ during fœtal life exert their power to arrest or prevent development at different periods, so that up to the time the power is experienced the heart maintains its normal form. He accordingly divided the subject into four classes as follows:

1. Those in which the retardation of development takes place at such an early period of fœtal life that the heart remains in a rudimentary form consisting of two or three chambers, primitive arterial trunks and imperfect aorta and pulmonary artery.

2. Those in which the development suffers interference at a later date when the auricles and ventricles are well advanced, but not necessarily perfect, and the aorta and pulmonary artery have almost, or entirely, reached completion. In these cases with defective separation of the ventricles and auricles there is constriction or total obliteration of the arterial or auriculo-ventricular passages with malposition of the aorta and pulmonary artery.

3. Those in which development is arrested in the advanced stages of fœtal life, when evolution of the auricles, ventricles, septum and great vessels has become completed, but defects remain which interfere with growth, the heart not undergoing the change which it should at birth. In such cases premature closure of the foramen

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* Comp. Rend. Séances et Memoires de la Société de Biologie, Ch. Féré, Paris, 1893–1895.

† J. W. Ballantyne, M.D., Pædiatrics, New York and London, 1896, vol. i.

ovale, deficiencies of the ductus arteriosus and obstruction at the auriculo-ventricular orifices are found.

4. Those in which there are deformities of the valves or vessels which, while not necessarily the immediate cause of disease, may prove at some future time to be the bases of various organic lesions and disease processes.*

Fœtal endocarditis probably is a more active factor in the causation of congenital anomalies, especially those of the valves and orifices, than is generally supposed. It leads to change in a manner similar to that which occurs in antenatal life, except that the seat of the process is usually in the right side of the heart. This is due to the greater activity of the fœtal circulation on that side of the organ and the flow of blood rich in oxygen from the placenta. The structural changes in the valves are of a slow sclerotic nature and are of various forms, thickening, contraction, fusion of the cusps, and induration and contraction of the chordæ tendineæ.

Anders calls attention to the small, round bodies, "nodules of Albini," normally present on the mitral and tricuspid valves which must not be mistaken for pathological vegetation.

Any of the valves may be affected, but in the greater proportion of cases of congenital endocarditis which live, the lesion is at the pulmonary orifice. Pulmonary stenosis is the most frequent valvular defect, and is the commonest form of congenital heart disease found in children who survive infancy. It is usually attended with stenosis of the conus arteriosus of the right ventricle and patency of both the ductus arteriosus and foramen ovale. In some cases complete stenosis of the pulmonary and tricuspid orifices occurs.

Obstruction and insufficiency may likewise be observed at the tricuspid orifice, and sometimes accompany similar conditions of the pulmonary orifice. Aortic lesions are rare and mitral more so, the latter is generally associated with tricuspid stenosis.

Heredity is an important feature in the development of cardiac anomalies and it is not uncommon to observe several infants in one family born with these defects. In such cases syphilis is sometimes an element.

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* T. B. Peacock, M.D., Dictionary of Medicine, Richard Quain, M.D., New York, 1883.

As regards the sex, male infants are more prone to cardiac malformations than females.

Symptoms.—Congenital anomalies of the heart are, as a rule, attended with striking phenomena. In rare and isolated cases certain abnormal conditions, however, such as perforate septum, may remain latent, being discovered only on post-mortem examination, sometimes after a life of many years.

The several forms of malformations, notwithstanding their variability, are attended with the same general symptoms. This is due to the fact that in spite of their anatomical differences they lead to similar results.

Of all the symptoms, cyanosis is the most prominent. From its intensity the expression morbus cœruleus, or blue disease, has been applied to the condition. The face, lips, tongue, as well as the extremities, are all of that dead bluish tint which indicates excess of carbonic acid in the blood. Crying or coughing greatly aggravates the condition. When the defect is slight it may not be apparent at birth or it may only be apparent when the infant cries. In older children any exertion greatly increases the blueness of the skin. Convulsions may attend coughing or exertion.

Breathlessness is another prominent symptom. It may be absent at first, but effort of any kind will cause it to be excessive. Clubbing of the fingers and toes with arching of the nails is commonly observed.

In addition to the bluish tint the countenance shows other departures from the normal; there is undue prominence of the lips and nostrils and dulness of expression. Ophthalmoscopic examination discovers some duskiness of the retina with tortuosities of the veins. Hæmorrhages from the mucous surfaces are not uncommon and occasionally there is a transudation into the subcutaneous tissues and serous cavities. The surface of the body is cold, especially that of the extremities. The patient also shows decided sensitiveness to cold. Extreme arching of the chest over the region of the heart is observed in some instances. In older children the mind is torpid and mental development slow. Such children are taught with difficulty. Notwithstanding the embarrassment of the circulation, dropsical symptoms are rarely, if ever, apparent.

While symptoms and signs characteristic of individual malformations are, as a rule, entirely absent, and differentiation impossible, there is a certain proportion of cases which sometimes presents special features.

Simple transposition of the heart to the right is not attended with any signs or symptoms other than transposition of the normal characteristics. Acardia and ectopia need scarcely be more than mentioned.

LESIONS OF THE PULMONARY VALVE—In the case of pulmonary obstruction there may be a small, empty pulse, a thrill over the base of the heart, enlargement of the area of cardiac dulness to the right, a loud, generally rough systolic murmur heard with its point of maximum intensity in the second intercostal space and widely propagated over the thorax anteriorly and posteriorly.

Pulmonary incompetence is usually associated with obstruction. Its presence may be recognized by a diastolic thrill at the base of the heart with its maximum intensity along the edge of the sternum about the level of the third intercostal space or fourth costal cartilage. This murmur is sometimes high pitched and not generally diffused. It is heard downward as far as the ensiform cartilage.

LESIONS OF THE RIGHT AURICULO-VENTRICULAR ORIFICE.—Tricuspid deficiencies are not as frequent nor are they attended with such marked symptoms of circulatory embarrassment as those of the pulmonary orifice. There is less cyanosis and breathlessness and often absence of clubbing of the fingers. The signs which are most noticeable are a heaving impulse in the epigastrium, increase in the area of cardiac dulness to the right and the presence of a murmur. If obstructive, it is presystolic with a distinct thrill in the region of the ensiform cartilage; if regurgitant, it is systolic.

LESIONS OF THE LEFT AURICULO-VENTRICULAR ORIFICE.—In some instances there may be congenital smallness of the aorta or of the arterial system, or fœtal endocarditis may affect the aortic valves. In these cases obstruction may occur attended with patency of the ductus arteriosus. The left ventricle may be hypertrophied. Mitral obstruction may also be observed.

Congenital contraction of the aorta is attended with a bruit, occasionally by a *fremissement cataire*. It is heard with its maximum degree of intensity at the base and in close proximity to the sternum. It is systolic in character and is accompanied with increase in the area of dulness from hypertrophy of the left ventricle. The pulse is hard and small.*

LESIONS OF THE AURICULAR SEPTUM.—A patent foramen ovale or other opening in the auricular septum may exist without causing cyanosis, provided valvular lesions are absent. Post-mortem examination has disclosed the presence of such anomalies in persons who have lived to advanced age.

In new-born infants, if the lungs are only partly expanded a portion of the blood which normally should flow into the pulmonary circulation passes directly from the right heart to the left through the foramen ovale. Bronchitis may have a decided effect on retarding the closure of the valve. There is always, however, a fair hope that if the child remains free from pulmonary complications the valve may close.

The foramen is usually open when there is stenosis of the pulmonary or tricuspid orifices. The murmur produced is audible at the base of the heart, both anteriorly and posteriorly.

Peculiar features may sometimes be observed, for example, if mitral insufficiency is present in addition, there may be venous pulsation in the neck, or there may be cerebral embolism caused by thrombosis of one of the femoral veins, the thrombus passing through the foramen ovale.

LESIONS OF THE VENTRICULAR SEPTUM.—Deficiencies of this nature are usually found associated with pulmonary obstruction or when the aorta arises from both ventricles, or where there is transposition of the great vessels.

The physical signs of ventricular septal defects are enlargement of the area of cardiac dulness to the right and a systolic murmur at the fourth left costal cartilage. The murmur has been described as loud and rough, replacing the first sound and heard with its greatest intensity over the lower part of the sternum. It is also heard in the axilla and posteriorly. But on account of the frequent association of other lesions, the signs peculiar to ventricular septal deformities are rarely observed.

PERSISTENCE OF THE DUCTUS ARTERIOSUS.—As a single lesion, this is extremely rare, but in association with other forms of congenital anomalies it is not uncommon.

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* Diseases of Children, Henry Ashby, M.D., and G. A. Wright, M.D., London, 1889.

The characteristic physical signs are dulness on percussion to the right, a fullness almost an inch from the border of the sternum in the second left intercostal space, a thrill on palpation and a murmur on auscultation in the same locality. The latter, however, is heard best posteriorly at the left of the spinal column on a level with the third or fourth dorsal vertebra.

The murmur is late systolic, high pitched, prolonged, and is louder on deep inspiration. There is also accentuation of the second pulmonic sound from increased blood-pressure in the pulmonary artery.

Some writers mention interference with the voice from pressure of an aneurysmal dilatation of the duct on the left recurrent laryngeal nerve.

Diagnosis.—Generally, there is no difficulty in recognizing the presence of congenital malformations of the heart. Persistent cyanosis aggravated by crying or coughing in the newly born infant does not leave much doubt. Differentiation of the lesion, however, is quite a different matter and practically it is frequently impossible, as distinctive physical signs are absent in the greater proportion of cases.

In those lesions of the heart in the adult which are of congenital origin, it is difficult or often impracticable to recognize the fact that the starting-point is a congenital defect. Such a source should be regarded as possible when there is disease of the tricuspid and pulmonary valves.

Prognosis.—The outlook is grave. The duration of life varies according to the degree and nature of the deformity. When the heart is in a more or less rudimentary condition life is possible for only a few hours, or, at most, a few days. In transposition of the aorta and pulmonary artery, the foramen ovale and ventricular septum remaining open, life may be prolonged for a few months. In mild defects patients may reach maturity or even advanced age.

Peacock gives the following as illustrating the possibility of viability. In constriction of the pulmonary artery without other abnormalities, cases are on record in which forty or sixty-three years were attained. When the foramen ovale was open and the pulmonary orifice decreased, one patient lived to forty and one to fifty-seven years of age. In the instance of defective ventricular septum nine patients lived to between twenty and thirty years of age, with patency of the ductus arteriosus, some lived from thirteen to nineteen years, but the greater proportion died much younger. Gibson, however, mentions two cases living to the age of twenty-three and twenty-six years respectively. With atresia of the pulmonary artery or orifice few live longer than two years.*

Treatment.—The management of congenital malformations which permit of viability is chiefly hygienic. The surface of the body should be kept warm, the diet nutritious and easily digested. In the case of infants, care should be taken to avoid exposure to any of the infectious diseases of childhood. In all instances when possible the patient should live in a dry, mild equable climate and in the open air as much as possible.

In the case of adults the same general line of treatment should be followed as in the instances of valvular disease.

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* T. B. Peacock, M.D., Dictionary of Medicine, Richard Quain, M.D., New York, 1883.

PART II.

DISEASES OF THE THORACIC AORTA.

DISEASES OF THE AORTA.

WHILE the aorta is subject to the same morbid processes as other arteries, its structure, the amount of blood-pressure to which it is subjected, and its anatomical relations lend especial importance to the consideration of the diseases which affect it. The coatings which compose the walls of the aorta, as in other vessels, are three in number, but differ in the following respects: The intima is much thicker from the presence of muscular cells, and more connective tissue and elastic fibre arranged in laminæ; the middle coat is for the most part made up of connective tissue and yellow elastic fibre, instead of unstriped muscular fibre, and the outer coat or adventitia is much thinner than that of the smaller vessels. These features render the aorta stronger and more elastic, at the same time less contractile.

From its relation to the heart it will be seen that it is subject to a great amount of stress from the blood-pressure, and that this is not equal, being greater at the greater curvature.

The diseases which affect the aorta are acute aortitis, chronic aortitis or arterio-sclerosis and atheroma and aneurysm. Invasion of the walls by pyogenic organisms, resulting in suppuration, may also occur, but is very rare. This condition is part of a general sepsis, and cannot be recognized during life. The aorta may also become involved by extension of ulcerative endocarditis, but by far the most frequent changes are those of arterio-sclerosis.

ACUTE AND SUBACUTE AORTITIS.

Acute aortitis is a rare disease. It may occur in the course of an acute general disease of which it forms an essential part, or independently, unaccompanied by evidences of any acute general affection. It is a matter of some doubt in the latter instance whether it ever arises primarily in a previously healthy aorta, as evidence points rather to its being an acute process engrafted upon a chronic inflammation.

Ætiology.—The causation is not always clear. Traumatism and extension of inflammation from neighboring structures, as the pericardium and mediastinal tissues are probably the more frequent sources. Acute infections, such as rheumatism, endocarditis, scarlet fever, small-pox and typhoid fever, have been followed by acute aortitis and furnish types of affections wherein it may constitute part of the general condition. Its association with influenza has been noted by Sansom and Fiessinger, and with tuberculosis by Huchard. Occurring independently of general disease, a prëexisting chronic aortitis is almost invariably present. In this form the source of the acute process is doubtless either endocarditis, pericarditis, pleurisy, pneumonia or nephritis. Pregnancy and parturition, it is said, have also been complicated with acute aortitis. The causes may be summed up as consisting of infections and toxicants.

Morbid Anatomy.—The aorta is generally enlarged, assuming a more or less fusiform appearance. There may be evidences of arterio-sclerosis except in cases arising in the course of acute general disease. On the inner surface of the aorta there are a number of translucent patches, bluish-white in color, and soft in structure in the earlier stages and yellowish-gray and firm in the later. Passing the fingers over the surface these patches will be observed to present slight elevations with intervening depressions. They arise from a fluid exudation into the intima, containing round and oval cells which also involve the elastic fibres of the media. The changes are successive, some appearing as recent formations, others as late. These changes are generally situated in the ascending and transverse portions of the aorta, leaving the aortic valve free, and may become the starting-points of chronic degeneration and undergo fatty metamorphosis and calcareous infiltration. They may also be situated at the juncture of the great arteries (carotid and innominate) with the aorta sometimes surrounding their orifices in a circlet, so as to encroach upon their lumena. Narrowing of the coronary arteries may likewise be observed from the inflammatory changes. Sometimes the process is confined to the neighborhood of these vessels.

Abrasions or ulcerations are occasionally found which may prove the source of embolism. In some cases the intima is blood-stained. This may be due to rupture of a nutrient artery or to post-mortem staining from the coloring matter of the blood. Not infrequently the patches may be stained almost black. The media and adventitia become thickened by round or flat-celled infiltration and lose their resiliency. They also show the presence of newly formed blood-vessels with intra-cellular hæmorrhages. In some cases the adjacent pericardial structure shares in the process constituting a peri-aortitis. A very important result of these changes is the implication of nerve filaments of the cardiac ganglia which surround the aorta, which may prove the source of angina pectoris, and in very rare instances of pleurisy.

Under the microscope the patches are seen to be made up of a small and large cell infiltration which involves the superficial layers of the internal coat. The middle coat is also infiltrated with cells.

Symptoms.—While there are a certain group of symptoms which may be said to characterize acute aortitis, in many instances the affection either remains latent, giving no positive evidence of its presence, or is totally obscured by the disease of which it constitutes a part. The latter is especially liable to be the case when associated with endocarditis, pneumonia and pleurisy, more especially the latter.

The symptoms which are said to characterize acute aortitis when the disease is manifest are dyspnœa of a somewhat peculiar type, in that inspiration is long and painful and expiration short. It is usually evidenced only by exertion, but occasionally may appear in paroxysms without effort. There also is a sense of weight and constriction in the præcordial region. Sometimes there is a sensation of a lump in the throat like globus hystericus. Generally, the patient is unable to assume a recumbent position. Cough is occasionally present. The præcordial distress may give place to severe pain which may be of the most agonizing character. It is described as a burning or tearing beneath the sternum, apparently in the region of the arch of the aorta, and is transmitted to the back down the spine and arm. Tenderness may be present to the left of the sternum in the first, second and third intercostal spaces. Vertigo may sometimes be experienced and persistent insomnia may prove a source of much distress. Dysphagia, which apparently has some relation to the proximity of the aorta to the œsophagus, is not uncommon. Symptoms of indigestion are generally present in most instances, being those of nausea, meteorism and sometimes vomiting.

The countenance of the patient with acute aortitis is expressive of great anxiety. There is usually paleness of yellowish or leaden hue. The extremities are cold and there may be slight pitting. As a rule, there is no elevation of temperature unless the affection is part of a general process attended with fever.

Forcible pulsation of the carotids is said by some observers to be present in certain cases. A more vigorous pulsation of the right subclavian than of the left has been noted, due, it has been suggested, to previous endocarditis. Potain has called attention to an upward displacement of the subclavian arteries, so that they can be felt in the hollow above the clavicle, this sign being more marked in the right subclavian. The cause of this phenomenon is doubtless due to the enlargement of the aorta and the consequent uplifting of its branches.

PHYSICAL SIGNS.—*Inspection* shows no change in the præcordial region unless the case is one which has followed a condition of arterio-sclerosis, when it is usual to observe the apex impulse displaced downwards and outwards. *Palpation* shows increase of intensity and prolongation of the impulse, if seen in the early stages; later the condition is one of weakness. *Percussion* in many instances reveals some increase in the area of dulness. *Auscultation* simply shows intensification of the second aortic sound. A soft systolic murmur may, however, accompany the first sound in the aortic area and a diastolic aortic murmur may develop in the more advanced stage.

Diagnosis.—Clinically, acute aortitis is seldom recognized during life, and the symptoms which are described as attending it, namely, pain in the aortic region, the signs of aortic disease and dyspnœa, belong to other conditions as well, and are in no way characteristic. Walshe suggests that pain, the presence of a thrilling pulsation in the course of the vessel, with an arterial murmur transmitted along the spine not attributable to a murmur of the aortic or mitral valves, indicates acute aortitis, but it is needless to say these conditions may be simulated by other affections except as to the arterial murmur. The presence of septic symptoms suggests suppurative aortitis, and of embolism, ulceration. Sudden cyanosis and death point to rupture of the aorta.

With such a limited clinical knowledge it is evident that the differential diagnosis must be more or less presumptive. The diseases which would seem most likly to be the source of confusion when acute aortitis is recognizable are angina pectoris and hysteria.

The pain of aortitis closely resembles that of true angina, but may be distinguished by its incessant character; it is remittent and paroxysmal, but never entirely ceases. From hysteria, aortitis may be differentiated by the persistent presence of the constriction pain in the aortic region, by the absence of any inclination to move about, which is especially more or less pronounced in hysterical conditions, and by the expression of the countenances which shows greater distress and a more decided pallor. The numerous protean symptoms of hysteria are also absent.

Prognosis.—Most patients with acute aortitis die, the disease being most always fatal in the primary acute forms, but less so when part of a general disease process.

Treatment.—Absolute rest in the semi-recumbent posture, with light diet, should be enjoined. When the pain is severe an ice or hot-water bag, according to the individual susceptibility of the patient and the effect obtained, may be applied, suspended by a cradle to avoid its weight. Counter-irritation in some cases has relieved.

The remedies which will suggest themselves are arsenicum, belladonna, bryonia, cactus, colchicum, gelsemium, kali carbonate, kalmia, spigelia, veratrum viride and the iodides; for the nervous phenomena the bromides and ignatia. For the insomnia, sulphonal, trional or chloralamid may be employed. Morphine, hypodermically, in doses of ⅛ to ¼ grain, should be given when the pain is severe and persistent.

If there are indications of failure of the heart, digitalis, strophanthus and caffein should be administered, but, as a rule. such remedies will otherwise prove harmful.

CHRONIC AORTITIS.

Chronic aortitis is an arterio-sclerotic inflammation and atheromatous degeneration of the aorta. It is by far the most important morbid process which invades the structures of that great vessel, as it forms the basis of a large proportion of the changes which take place within its walls, with results that are both far-reaching and profound.

Ætiology.—In seeking the causation of chronic aortitis, one must look to the conditions which give rise to both general and local arterio-sclerosis. The disposition among certain families to affections dependent upon arterio-sclerosis has been repeatedly demonstrated. Thus heredity must be recognized as an important element. Again, men are more liable than women, not that there is any inherent tendency in the sex, but that males from the habits and duties of life are naturally more exposed to the causal influences.

Arterio-sclerosis belongs essentially to advanced years, and is often regarded as a senile change. In the instance of the aorta, while these changes are frequent in old age, they are also particularly liable to be encountered in early middle life, especially in able-bodied men. Occasionally, the process occurs in the young.

The general causes are numerous and somewhat varied. In the first place, there are the diatheses, notably the arthritic. The influence of gout has long been recognized in all conditions of arterio-sclerotic degenerations, and it is probable that excess of uric acid in the blood is one of the most potent causes. Predisposition to this condition is certainly heredity. When the aorta is affected by gout the aortitis is prone to be seated in that portion of the vessel which is just above the valves, and thus the orifices of the coronary arteries are very liable to become involved.

Syphilis is an important element in chronic aortic disease. Its course is obscure. Generally, it does not attack the aorta until long after infection. In some instances it may be hereditary, and this may account for chronic aortitis in the uncommon instances when it occurs in the young.

Diabetes, lead-poisoning and malaria may also prove the source of chronic aortic degeneration, but less frequently.

Excessive use of alcoholic stimulants is an important cause. It acts as a direct excitant of the circulation and as a blood irritant, thus increasing arterial tension.

Excessive muscular exertion, especially hard and prolonged laborious vocations, may also prove to be the cause of disease of the aorta. It is a factor in strong men in early middle life and one of the most important elements in chronic aortic disease.

Over-eating and drinking, by distending the vessel, is an active cause. The excessive use of tobacco may likewise give rise to aortitis by increasing arterial tension, on account of its toxic influence upon the nervous-system.

Bright's disease possesses a double relation to chronic inflammation of the aorta. It is first a distinct and positive cause; and, second, it may arise as the result of a general arterio-sclerosis.

In summarizing the various ætiological influences which are the source of chronic aortitis, it will be observed that they act as chronic toxicants of the blood, which cause either increased arterial tension, or which operate directly in raising the blood-pressure. Frequently, both these elements act conjointly.

Morbid Anatomy.—There is almost always some loss of the natural curve of the vessel, with dilatation and frequently small bulgings above the aortic valve. The lesion may either be confined to the aorta itself or may form part of a general arterio-sclerotic process. The latter is more usual. On laying open the vessel, if the process is in the earlier stages, a number of small whitish-gray, gelatinous-looking patches will be observed studded over its surface. These are somewhat elevated, presenting the appearance of a button, smooth and glistening, being yet covered with the normal lining of the vessel. In more advanced conditions they are roughened from loss of endothelia and yellowish and cloudy. These patches have been compared by Morgagni to a deposit of melted wax on a cold surface. They show a disposition to erosion from loss of endothelia and to the formation of calcareous deposits. The eroded surfaces form atheromatous ulcers which are yellowish in color and are composed of fatty material. These changes may be scattered throughout the extent of the aorta, but are more frequent at the ascending portion of the arch, particularly close to the aortic orifice. They are less frequent in the ascending and abdominal portions. A very important feature is the tendency to implication of the mouths of the coronary arteries, thus leading to the conditions which characterize endarteritis of the vessels, namely, angina pectoris, fibro-myocarditis and fatty degenera-

When the process is general the radial pulse, in addition to the probable presence of hardness, shows fullness and a high degree of tension. The pulse-wave is usually well sustained, though not large. There may be a sensation as if the artery rose from its bed. Occasionally, there may be a decided difference between the radial pulse of the right and left side, owing to the involvement of the orifice of one or the other vessel. When the process is confined to the aorta there may be no evidence of change manifested in the radial pulse.

While the clinical events above mentioned are mostly those which are directly associated with chronic inflammation of the aorta itself, the consequence of the process and the accompanying phenomena may include nearly all the conditions associated with degeneration of the cardio-vascular system.

PHYSICAL SIGNS.—*Inspection* may show the apex impulse displaced to the left and more diffuse than normal.

Palpation shows a heaving impulse similar to that of hypertrophy, unless the heart itself has undergone change.

Percussion discloses increase of the area of dulness, especially toward the left, and, if there is any degree of aortic dilatation, to the right of the sternum from the third intercostal space upward.

Auscultation shows the second sound to be slightly ringing and sonorous. It has been called by some writers musical. The first sound may remain normal; frequently, however, it is weakened and may be accompanied by a murmur. The aortic second sound is accentuated.

Diagnosis.—While in a large proportion of cases distinctive symptoms are manifest and the diagnosis is unattended with difficulty, there are many instances where it is impossible. This, of necessity, is the case in those forms of the disease which remain latent. It is also frequently impossible to recognize the affection when entirely local, for example, when situated in the ascending portion of the aorta at a distance above the valves, even when there are calcareous deposits, as in conditions of senility, or when limited to a small area just above the valves.

Prognosis.—Chronic aortitis is a steadily progressive disease. It may, however, be retarded in its progress. While it often leads to the gravest consequences and to conditions which end in death, as well as the normal cells break down in the fatty process and become transformed into oil globules and cholesterin crystals. The starting-point of this degenerative process, according to Hippolyte Martin, is due to endarteritis of the vasa vasorum, whereby obstruction of their lumen follows with consequent arrest of the nutrition of the tissues which they supply. As the transformed and softened material accumulates in the vacuoles, it presses forward until the endothelial breaks down and an atheromatous ulcer is formed. The softened material has been likened to gruel, from whence the term atheroma is taken. Associated with the patch there may be a deposition of salts, which are chiefly found in the middle coat, and which form hard calcareous plates, so that the aorta may be converted into a hard brittle tube. The deposits are not generally uniform, but irregular and broken, with sometimes projecting points which are favorable to the formation of coagula. In the later stages all the coats of the artery are involved, the intima is infiltrated, degenerated and eroded; the media is likewise degenerated and the adventitia dilated, its fibrous tissue thickened and its vessels dilated.

Implication of the coronary arteries occurs through either the direct effect of encroachment of the atheromatous or calcareous infiltration on their lumena, or by changes in the tissues of the aortic cusp, which may be displaced in such a manner as to obstruct the vessel. In many cases the coronary arteries themselves are the seat of the arterio-sclerotic process.

Symptoms.—Frequently chronic inflammation of the aorta remains latent, and its presence is not recognized until post-mortem examination. On the other hand, it may be attended by distinctive symptoms. These are commonly constrictive præcordial distress, dyspnœa and paroxysmal cardiac asthma. Sometimes there is cough and sometimes vertigo. There may also be substernal pain, which has a tendency to radiate down the arms and back, and which may be accompanied by a distressing sense of impending death. Not infrequently there are evidences of arterio-sclerosis of the superficial arteries, such as the temporals and radials, which are rigid and tortuous, feeling like whipcords. Excessive pulsation of the carotids and the subclavian arteries may be noted, pulsations of the latter being appreciable in the jugular fossa.

When the process is general the radial pulse, in addition to the probable presence of hardness, shows fullness and a high degree of tension. The pulse-wave is usually well sustained, though not large. There may be a sensation as if the artery rose from its bed. Occasionally, there may be a decided difference between the radial pulse of the right and left side, owing to the involvement of the orifice of one or the other vessel. When the process is confined to the aorta there may be no evidence of change manifested in the radial pulse.

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PHYSICAL SIGNS.—*Inspection* may show the apex impulse displaced to the left and more diffuse than normal.

Palpation shows a heaving impulse similar to that of hypertrophy, unless the heart itself has undergone change.

Percussion discloses increase of the area of dulness, especially toward the left, and, if there is any degree of aortic dilatation, to the right of the sternum from the third intercostal space upward.

Auscultation shows the second sound to be slightly ringing and sonorous. It has been called by some writers musical. The first sound may remain normal; frequently, however, it is weakened and may be accompanied by a murmur. The aortic second sound is accentuated.

Diagnosis.—While in a large proportion of cases distinctive symptoms are manifest and the diagnosis is unattended with difficulty, there are many instances where it is impossible. This, of necessity, is the case in those forms of the disease which remain latent. It is also frequently impossible to recognize the affection when entirely local, for example, when situated in the ascending portion of the aorta at a distance above the valves, even when there are calcareous deposits, as in conditions of senility, or when limited to a small area just above the valves.

Prognosis.—Chronic aortitis is a steadily progressive disease. It may, however, be retarded in its progress. While it often leads to the gravest consequences and to conditions which end in death,

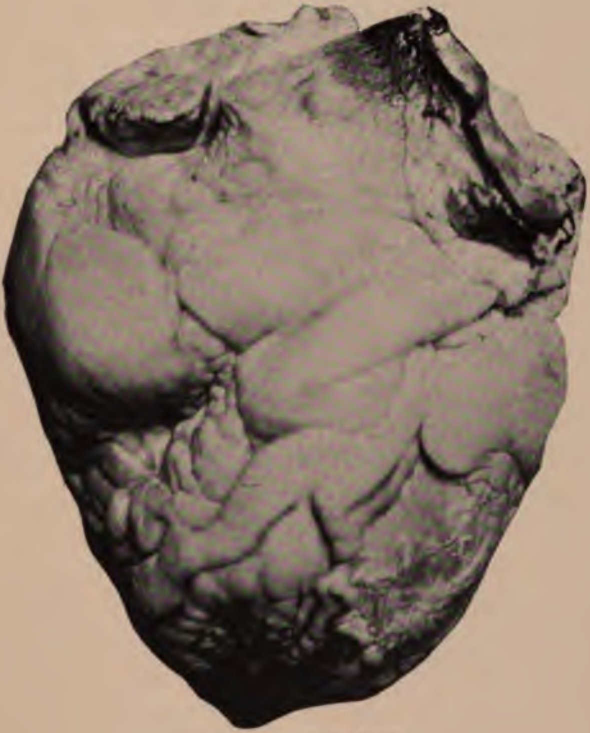

FIG. 34.—Fatty heart.

it does not always do so. The general condition of the patient, his habits, surroundings and circumstances of life, all exert an important bearing upon the outlook, but the most potent factor in the prognosis is the state of the heart, whether or not the sclerotic process has in any way affected it, or the coronary arteries are involved. In some instances death may suddenly occur from rupture, even in cases where the disease had not been suspected.

Treatment.—The treatment of chronic aortitis is mainly dietary and hygienic, and is substantially that of arterio-sclerosis. Rest, fresh air, nutritious food, avoidance of stimulants and the carbohydrates, and a limited supply of white meats should be the general line of diet, which in many respects is practically that prescribed for gout. Milk should form an important item of diet daily. A course each year at some suitable alkaline spring is commended.

Remedies are of use in retarding the progress of the disease, notably the iodides. Five-grain doses of potassium iodide may be given daily and continued for some time. The chloride of gold and soda may also prove useful. Glonoin is indicated in conditions of high-pulse tension. Arsenic is also a remedy of importance. When the heart structure becomes involved or angina pectoris appears, the special remedies for these conditions will be called for.

ANEURYSM OF THE AORTA.

Aneurysm of the aorta is a localized bulging of its walls filled with blood, which may act as a tumor pressing upon adjacent structures.

Ætiology.—The majority of aneurysms of the aorta are traceable directly or indirectly to chronic aortitis or arterio-sclerosis of the aorta. It therefore follows that the influences which underlie these disease processes must of necessity also be the causes of aortic aneurysm. They cannot, however, be considered as bearing the same ætiological relation, as some exert a much more marked influence in aneurysm and are in consequence of much greater importance in this connection. Again, while post-mortem examination demonstrates that aneurysms of the aorta are generally the seat of atheromatous degeneration, it also shows that atheroma of the aorta is much more frequently found without aneurysm, that is, the proclivity to aneurysm is not co-equal with that to atheroma.

Aneurysm of the aorta shows a decided tendency to occur at a certain period of life and is more frequent at about forty years of age, the average limits being between thirty and fifty-five. The former, it will be observed, is somewhat early for atheromatous degeneration. This is explained by the fact that arterio-sclerosis becomes more active in the presence of increased vascular tension and that vigorous persons, especially men, who may be suffering from its earlier inroads, by muscular exertion and strain, enhance its influence. Thus it seems that aneurysm occurs at that period of life when the individual is still strong and exposed to muscular strain and yet at the same time is liable to arterio-sclerosis.

Men are very much more prone to the affection than women, the proportion being, according to some statisticians, about nine to one. This is obviously due to the different influences which surround the two sexes and not to sex as a factor, for, according to Coats, while aortic aneurysm is, as above stated, very much more frequent in men than in women, cerebral aneurysm is about equal in both sexes.

The causes which especially favor aortic aneurysm are syphilis, alcohol, gout, rheumatism and muscular strain. Syphilis operates in a decided way and is responsible for a very large proportion of cases, more, in fact, than any other one cause. Aitken found that in a number of cases of aortic aneurysm among soldiers 50 per cent. followed syphilitic infection. Other observers give much higher figures. Syphilis of the aorta may assume a gummatous form similar to gumma in the walls of the heart, or it may appear as an ordinary arterio-sclerosis. Alcohol, gout and rheumatism are active causes, but to a much more limited degree.

The relation of muscular strain to the causation of aneurysm is one of importance. It may be said to be twofold. In the first place, laborious occupations requiring excessive muscular effort by constantly raising the arterial tension is probably the most active determining cause. It has been noted that porters, stevedores, hammerers and other mechanics who lift heavy weights are especially prone to aortic aneurysm. It has also been found by British writers to be much more prevalent among soldiers than among

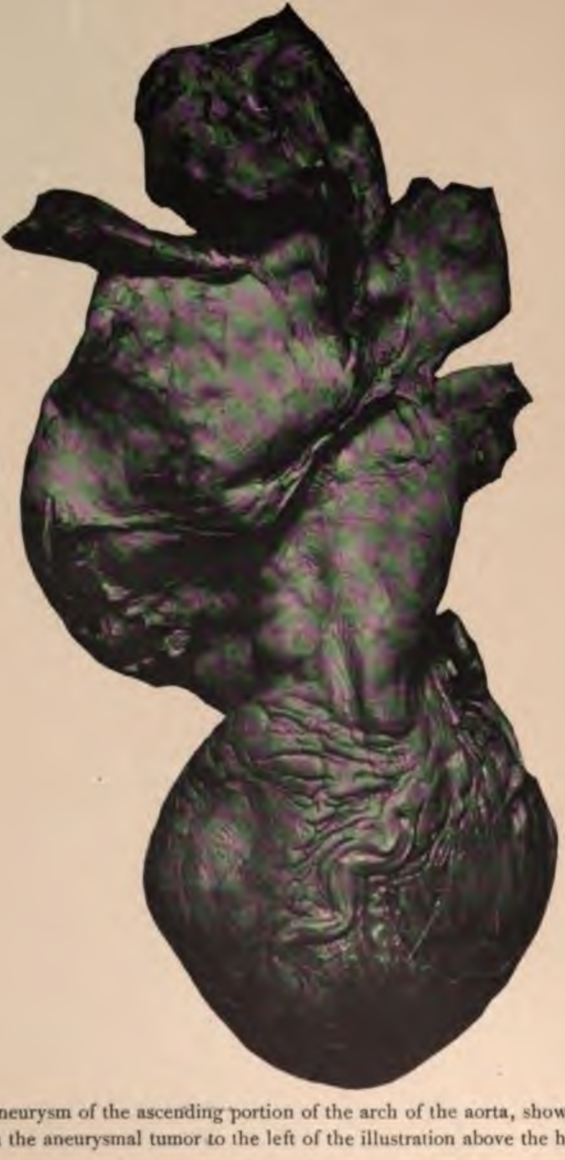

FIG. 36.—Aneurysm of the ascending portion of the arch of the aorta, showing the aorta with the aneurysmal tumor to the left of the illustration above the heart.

civilians, the cause being assigned to the strain arising from the long marches, carrying heavy accoutrements and knapsacks.

Again, sudden violent muscular strain may be the starting-point of an aortic aneurysm, but it is a matter of some doubt whether severe exertion alone can actually rupture a previously healthly aorta. This is, in fact, denied by many observers. It is probable, however, that rupture of some of the branches of the vasa vasorum may take place in this way and prove the starting-point of an arterio-sclerotic process.

On the other hand, the onset of not a few cases is frequently directly attributable to muscular exertion. It is not uncommon for the patient to state that he felt something suddenly give way. This is really what takes place, but in a diseased aorta. The media becoming lacerated by the extra strain put upon the walls, not yet being strengthened by the compensatory thickening over the patch of meso-arteritis, gives way or an advanced atheromatous area dilates. Under these conditions the degree of muscular strain which may weaken the aortic wall is sometimes very slight.

Traumatism in the form of direct violence, such as heavy blows on the chest, has been known to be the cause of some aneurysms.

Morbid Anatomy.—Aneurysms of the aorta in 75 per cent. of all cases are situated in the thoracic portion, of which over one-half are in the ascending portion and the remainder about equally divided between the descending and transverse portions of the arch. In some instances aneurysm may commence above the sinuses of Valsalva and attain a quite large size. Aneurysm within the pericardial sac may also occur. They are generally small and liable to rupture early. The anterior walls of the aorta are usually the seat of aneurysmal process except when the descending portion is involved, when the tumor is generally situated posteriorly. The direction of bulging, however, is governed by the relation of the weakened part of the wall to the situation of the point of strongest impingement of the blood-current.

The seat of aneurysm of the aorta according to frequency may be summarized as follows:

(1) The beginning of the aorta as it emerges behind the trunk of the pulmonary artery. In this situation aneurysm may give rise to hypertrophy of the right ventricle early on account of the resistance

to the outward flow of blood through the vessel, causing a basic murmur in the pulmonary area and relative insufficiency of the tricuspid valve.

(2) The ascending portion behind the sternum at the junction of its manubrial and gladiolar sections. At this point the aneurysm may erode the bone and push directly against the integument.

(3) The convexity of the arch toward the apex of the right lung. The pleural cavity at this locality may become obliterated by an inflammatory process and the aneurysm may perforate the lung tissue, rupturing into the bronchioles of the apex with fatal hæmorrhage.

(4) Between the innominate and left carotid at the apex of the arch behind the trachea. In this situation it may perforate into the trachea before becoming very large.

(5) Posteriorly in the descending portion, between the left subclavian and the isthmus of the aorta to the left of the vertebral column. In this situation aneurysms are usually fusiform.

Aneurysms in other localities generally all tend to point toward the vertebral column, but the resistance here encountered causes them to assume a lateral direction. These are liable to rupture into the pleural or abdominal cavity. Aneurysm of the thoracic aorta may dissect its way between the posterior muscles of the trunk and may attain a large size without rupture.

As in other arteries, aortic aneurysm may be sacculated or fusiform. The first-named variety is that which is generally considered in the following discussion, and which is implied when speaking of aneurysm of the aorta without qualification.

Sacculated aneurysm, which is the type most frequently encountered, varies all the way in size from that of a pea to a sac as large as the head of an adult. In some instances there may be a number of separate dilatations or tumors. The starting-point is a giving way of the media. A portion previously weakened by disease under the stress of the blood-pressure, perhaps with some additional strain, bulges outward. The walls of the sac are always formed of the arterial coats, at least in part. There is a decided variation in the extent to which the latter participate in the same. The intima is present always to a greater or less extent and can be generally traced from the border of the opening into the sac for some distance inward. In the smaller aneurysms it may line the entire interior, but in the larger it extends only a short distance. The media, as may be expected from its undergoing previous degeneration, disappears generally near the border of the sac. The adventitia or outer coat, which in the aorta is normally thinner than in other arteries, becomes very much thickened and forms the principal part of the sac wall. It shows increased vascularity and connective tissue with a decided tendency to adhere to adjacent structures from inflammatory action. The fibrous tissue may also proliferate in and around the neighboring tissues and form part of the external coat. The orifice connecting the aneurysmal sac with the aorta displays all degrees of size, varying from a few lines to several inches; when narrow there is more likelihood of a bruit. The interior of the sac is usually found to present a roughened surface with frequently several diverticula or pouches and contains more or less fluid blood. It is oftentimes covered with a layer of coagula, the outer being grayish-white, sometimes quite white from loss of blood pigment, the inner dark red from excess of blood stain. Sometimes the different layers of the vessel are stained irregularly in patches from blood which has forced itself in between. The phenomenon of the formation of the clot in sacculated aneurysm consists, first, of the adhesion of the white blood-corpuscles to the morbidly roughened walls of the sac, then a chemical process commences which is generally explained as due to the action of a fibrin-ferment in the blood arising from the blood-platelet (a special variety of white blood-corpuscle) upon the fibrogen. This ferment is not present except when the blood-platelets have undergone disintegration.

Another explanation is that of Woolridge, namely, that a substance in the white blood-corpuscle called lecithin, in union with fatty acids, is the cause of the coagulation.

The walls of the aorta show arterio-sclerotic and atheromatous changes as described in chronic aortitis. In some cases they may, except for the aneurysm, appear healthy. This, however, is very uncommon.

In many instances the cusps of the aortic valve reveal evidences of involvement, generally of a sclerotic nature, obviously arising from the same causes which are the source of the process in the aorta itself. The arterial trunks emerging from the aorta very frequently suffer interference by obstruction of their lumena by the aneurysmal tumor or by atheroma or coagula.

Fusiform aneurysm is most frequent in the ascending and transverse portions of the arch. Generally, the whole circumference of the wall is involved, but there may be decided irregularity. The arterial trunks which branch off from the aorta are also generally dilated. In this respect fusiform aneurysm differs from sacculated aneurysm which less frequently involves the branches. There is no special reason to suppose that the pathological process which gives rise to fusiform aneurysm is materially different from that which attends the sacculated variety. The walls of the vessel become weakened from degeneration and dilate from the force of the blood-pressure, but the morbid process is general, hence the dilatation is of that character instead of pouch-like.

Examination shows that the walls are thinned in some areas and thickened in others and that the newly-formed tissue undergoes fatty or calcareous degeneration. All the coats are implicated; the intima shows arterio-sclerosis, the media diminution in thickness, and the adventitia increase in thickness. The tendency to coagulation of the blood is very much less than in the sacculated variety.

A feature of fusiform aneurysm is its tendency to remain stationary and the absence of symptoms even after physical signs are apparent.

Dissecting aneurysms are usually found in the arteries of the extremities, but may occur in the aorta. The intima ruptures and allows the blood to force its way between its walls and those of the media or between the layers of the media. The blood may burrow for a long distance and sometimes force a second opening into the aorta or rupture the adventitia.

The effect of aneurysm of the aorta on the adjacent structures is frequently most marked. It almost always causes displacement of adjacent organs. The heart, lungs, œsophagus, bronchi and mediastinal tissues may all suffer in this way. While the heart is frequently displaced, it is not necessarily hypertrophied or dilated. Hypertrophy follows when the aortic valves are involved; otherwise a large aneurysm may or may not be accompanied by a small heart.

In some instances aneurysm may form a cavity in the upper portion of the ventricular septum and communicate by false openings with the pulmonary artery and left ventricle or may force its way between the visceral and parietal layers of the pericardium and displace the heart backward. It may also perforate into the right auricle or left ventricle or superior vena cava.

The neighboring organs and tissues also undergo changes in the form of inflammatory processes with resulting adhesions and structural changes from pressure. Thus a hollow viscus may be changed in both structure and size. Pressure on the lung may occlude a bronchus and thus cause collapse of the corresponding lung tissue. The change in the lung from pressure of aortic aneurysm frequently gives it an appearance like the spleen. Pressure upon the bones frequently causes their entire destruction by erosion. An aneurysm may thus bore its way entirely through the sternum or ribs, and completely obliterate the osseous structures. The vertebræ are liable also to undergo this destructive process, but to a less degree, on account of their greater strength and the tendency of the aneurysmal tumor to press forward.

Symptoms.—The symptoms of aneurysm of the aorta are those which for the most part arise from the effects of pressure. They vary according to the size and location of the tumor. Yet in some instances an aneurysm may attain a considerable bulk and afford no evidences of its presence until fatal rupture or autopsy reveals it, or it may, on the other hand, give rise to more or less vague symptoms of difficult determination. With such positive and negative characteristics aneurysm has been variously classified by different writers. The most comprehensive grouping is as follows: (1) Those in which there are no symptoms; (2) those in which there are symptoms of pressure of an indefinite character, yet the physical signs are wanting; (3) those in which the signs and symptoms are more or less clearly defined.

In the first group the tumor is usually small. Those seated close to the sinus of Valsalva are frequently of this variety. In the instance of the second group there are always at least some pressure symptoms present.

The most common and striking clinical features in characteristic cases are dyspnœa, pain, the presence of a pulsatile tumor and præcordial distress.

Aneurysm involving the transverse aorta, owing to the limited space for expansion, is prone to be attended with pronounced symptoms at an earlier period than when situated in other parts of the aorta. Interference with the function of respiration is frequent and may arise from direct pressure or its indirect effects. In the instance of large tumors breathlessness is almost invariably a constant symptom. It is also prominent with those of smaller size when situated in unfavorable localities. Pressure upon the lung may not only reduce the breathing surface, but may also give rise to secondary changes. Direct pressure may also affect the trachea or obstruct the pulmonary artery and pulmonary vein, and through the stasis thus induced may prove a persistent source of dyspnœa. The irritation and weight of the tumor as it presses upon the pneumogastric or recurrent laryngeal nerves are the chief sources of the indirect influences of aneurysm on the respiratory function. In these instances the dyspnœa may be paroxysmal.

Pain is generally a very prominent symptom, especially in deep-seated tumors. It is usually of a gnawing, boring or burning character, due to the severe pressure of the tumor on the surrounding structures, especially the vertebral and the intercostal nerves. The pain arising from pressure on the vertebral column is sometimes most intense and may throw the patient into a state of frenzy. Anginal attacks also occur, particularly when the aneurysm is situated near the heart. On the other hand, pain may be entirely absent even in the presence of severe erosion of the bone. Such instances, however, are rare.

The direction in which the pain radiates is of some diagnostic significance. Radiation down the left arm and to the neck and occiput is an accompaniment of aneurysm of the transverse portion; pain referable to the intercostal nerves points to aneurysm of the descending aorta; when situated just above the diaphragm, to aneurysm in that locality. Pain is, however, not confined to these areas, but may run down both shoulders and arms.

Præcordial distress, such as palpitation, fluttering, etc., is almost always constant and in some cases is the first symptom to draw attention to the fact that some circulatory disturbance is present. Sensations of præcordial weight, soreness, constriction, and neural pains in various areas from pressure are common.

A pulsatile tumor, while an important symptom and one which is decidedly characteristic, is by no means constant, at least at the time the patient is first observed. In many instances, however, it develops later. The growth of the tumor is sometimes very rapid and the size which it may attain very large. The integument covering it may be stretched, smooth and shiny. In advanced stages it may be inflamed and even gangrenous.

In addition to dyspnœa, pressure on the pneumogastric nerve is the source of various disturbances of the alimentary canal, especially nausea and vomiting. It may also cause asthmatic attacks.

The mechanical effects of the weight of the tumor on the recurrent laryngeal nerve may produce changes in the quality of the voice, such as hoarseness and aphonia. In the earlier periods, spasm of the cords may occur with stridulous breathing. Laryngoscopic examination may show the cord on the affected side to be paralyzed. The adductors, especially the left, are usually the muscles first affected. Unilateral paralysis of the diaphragm may be present from pressure on the phrenic nerve. This is liable to happen on the left side when the descending aorta is the seat of lesion.

Cough is common and may be due either to implication of the vagus or recurrent laryngeal nerve or to the direct effect of pressure upon the trachea. Aneurysm of the transverse portion of the aorta may give rise to a peculiarly harsh, strident cough. When the aneurysm is situated in the descending portion it may press upon the left bronchus, in which case there is a persistent cough, but without the peculiar harshness of that which accompanies aneurysm of the transverse portion. In these cases of pressure on the trachea and bronchi a bronchitis may develop attended with abundant expectoration, watery at first and later thick and opaque. Hæmorrhages may attend the cough, due either to severe congestion or to oozing from granulations on the mucous membrane of the trachea which arise from the pressure upon it.

Pressure upon the sympathetic nerve above the cilio-spinal ganglia causes changes in the pupil of a varying character on the affected side. In the earlier periods the pupil is dilated and its size may constantly vary. Later, when the function of the nerve has become either destroyed or impaired, it becomes contracted. Accompanying the dilatation there may be unilateral pallor, while with contraction there may be unilateral flushing and sweatings. These phenomena may arise with aneurysm in any part of the aorta.

Dysphagia is frequently present, arising from pressure upon the descending portion upon the œsophagus, or from reflex action from aneurysm in the transverse portion of the arch. The latter is indicated by paroxysmal dysphagia and is less frequent. In both instances nutrition may become impaired.

Venous stasis with œdema and sometimes cyanosis often results from pressure on the great veins, especially the innominate and the superior vena cava. Headache and various cerebral symptoms appear from the same cause. Less frequently œdema of the lower extremities arises from pressure on the inferior vena cava. Pressure of the vena azygos is attended with cyanosis of the lower portion of the thorax.

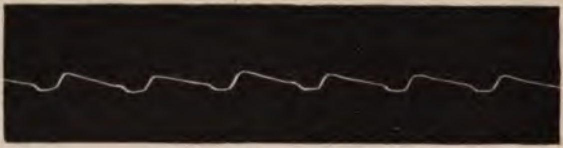

FIG. 37.—Right radial pulse.

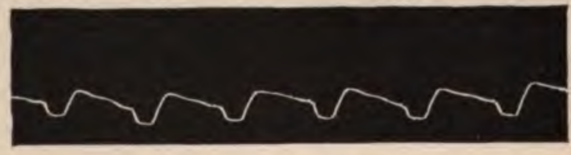

FIG. 38.—Left radial pulse.

Sphygmographic tracings of the radial pulse of a case of aneurysm of the ascending and transverse portions of the arch of the aorta. Metropolitan Hospital.

The right and left radial pulse show absence of perfect equality and synchronism, as shown in Figs. 37 and 38. Difference in volume is more especially associated with pressure upon the larger arterial trunks. The walls of the radials are generally rigid, but may be soft and yielding. The blood-pressure is variable and the rate usually slower than normal and rhythm regular. Sometimes the radial pulse is quite behind the systole of the heart. There are, however, many individual peculiarities.

Among other circulatory phenomena is pulsation of the arteries of the retina, which in one recorded case is said to have been more distinct on the left side than on the right.

The hæmopoietetic system does not usually suffer much interference, but pressure on the thoracic duct by aneurysm of the descending aorta may occur. The result is malnutrition and emaciation. Pressure upon the semilunar ganglia has been attended with pigmentation of the skin, as in Addison's disease, without œdema or change in the suprarenal capsules.

Clubbing of the finger-nails may sometimes be observed with aortic aneurysm.

General enfeeblement with evidences of slowly progressive loss of weight and exhaustion from the pain and great distress which often attend thoracic aneurysm may ultimately develop.

The local symptoms of thoracic aneurysm vary according to site as follows:

Aneurysm of the Ascending Portion of the Arch.—Greater prominence of pain, especially the presence of angina pectoris, dulness to the right of the manubrium, displacement of the heart downward and to the left, delay of pulse in the peripheral arteries, pressure symptoms pointing to implication of the sympathetic, superior vena cava and pulmonary artery causing a pulmonic systolic murmur.

Along the convex border of the ascending portion of the arch is the favorite site of aneurysm. In this locality it may grow very large and cause pronounced symptoms. In many instances a distinct pulsating tumor is present to the right of the sternum in the second and third intercostal spaces, the sternum itself sometimes forming part of the prominence. The tumor may cause most extensive changes, eroding the ribs by pressure outward, at the same time pressing upon the superior vena cava, causing œdema of the hand and arm, or when the subclavian alone is compressed œdema of the right arm only. When the bulk is excessive the heart is displaced downward and to the left. Rupture is liable to occur into the superior vena cava.

When situated immediately above the sinuses of Valsalva, aneurysm is frequently small and latent, being of that type to which allusion has been made which gives no manifestation of its presence until sudden death by rupture occurs which usually takes place into the pericardium.

In rare instances aneurysm is seated on the concave surface of the arch and presses backward and inward or occasionally presents itself as a tumor to the left of the sternum. A large growth in this locality may be the source of displacement of the heart downward and to the left. It may also press upon the recurrent laryngeal nerve and give rise to changes in the voice and respiration, or upon the inferior vena cava causing ascites and swelling of the feet. If there is an obstruction of the innominate artery the right pulse may be smaller in volume and even somewhat slower than the left. Œdema of the right side of the face and of the right arm may also appear. Pain down the right arm and contraction of the right pupil are common. Aneurysm in this part of the arch, in addition to rupturing into the pericardial sac, may also perforate the pleura, or by means of adhesions break into the lung itself. In rare instances it may rupture externally.

Aneurysm of the Transverse Portion of the Arch.—The tumor as it enlarges generally inclines backward. It may become very large, push forward and erode the sternum. Even when small and giving no physical signs it may cause many pressure symptoms from compression of the œsophagus, trachea and left recurrent laryngeal nerve, giving rise to dysphagia, dyspnœa, stridulous breathing and a strident suffocative cough, sometimes described as brassy. A small aneurysm in the posterior or lower portion of the arch may press upon a bronchus and cause bronchial and pulmonary changes, such as bronchitis, bronchiectasis and suppuration and breaking-down of the lung itself.

When the tumors are large and present themselves externally, it is usually to the right of the sternum or in the middle line, less frequently to the left. In some instances large aneurysmal tumors in this position may grow between the pleuræ involving the space between the manubrium and the vertebræ, the tumor being manifest in the sternal notch. Pressure on the innominate artery, less frequently the subclavian or carotid, sympathetic nerve and sometimes the thoracic duct may occur.

In some cases both the ascending and transverse portions of the arch are involved together. The direction of the tumor is then upward and to the right. When the innominate artery is not involved the pulse of the right arm and neck is normal ; that of the left is weak and tardy.

Aneurysm of the Descending Portion of the Arch.—In this situation the symptoms are often indefinite and sometimes are absent. The direction of the growth of the tumor which may be of large size and apparent to the left of the vertebral column opposite the angle of the scapula or below is to the left and backward. The dorsal vertebræ from the third to the sixth may become eroded, excessive pain attending the process and sometimes paralysis from pressure on the cord. Compression of the bronchi or lung, with consequent changes and dysphagia, often attend aneurysm in this locality.

Another feature which may sometimes be observed is smallness of the crural pulse in comparison to the radial. Intercostal pressure pains are common, and in rare cases the aneurysm may press forward and displace the heart or even press upon the œsophagus, into which it may break.

PHYSICAL SIGNS.—The first essential in examining the patient is a good light. In cases where the signs are not well marked, the eyes of the observer, in order to detect the slightest pulsation, should be brought close to the patient's chest. While in many instances the external changes are pronounced, in others they may be absent or slight. When the aneurysm is small and situated some distance from the walls of the thorax, it generally fails to give any physical signs. Pressure symptoms, however, may be present. When the ascending aorta and first portion of the arch are the seat of the aneurysmal process, physical signs are in evidence; when in the last portion of the arch and descending aorta, pressure symptoms alone may be recognizable.

Inspection, if not negative, reveals deformity of the chest wall, due to the presence of the tumor and to restriction of the lung and displacement of the heart. The most positive sign usually first observed is the presence of an abnormal diffuse heaving pulsation, either with or without an external tumor, which varies in position according to the part of the aorta involved; when to the right of the sternum, on the level of the third rib or in the first and second intercostal spaces, it points to the ascending aorta as the seat of the process; when to the left of the sternum it suggests the transverse portion of the arch, when posteriorly to the left of the spine in the dorsal region, to the descending aorta.

When a tumor is present it may occupy any of the above positions, the most frequent being that first mentioned, as the ascending portion of arch most likely to be affected. The costal cartilages on both sides and the sternum may become entirely obliterated, the tumor pushing through the upper part of the latter, sometimes forming a projection several inches in diameter. The integument covering it may be varicose, inflamed and blood stained, and in rare cases even gangrenous, as mentioned. The pulsations usually correspond to the systole of the heart and are expansile. They may be accompanied by a thrill. When the aneurysm is in the transverse portion the thrill may be felt in the suprasternal notch. The presence of pulsation in the neck denotes implication of the innominate artery. The degree of pulsation is influenced by the presence of clots in the sac, being more pronounced when they are few. When the movement is slight simple devices are often used to detect the pulsation. One plan is to fasten by means of plaster cones of paper to opposite sides of the tumor or area of suspected pulsation and observe whether they diverge; if there is pulsation they will do so. Another plan is to place two bits of paper edge to edge closely over the tumor; if they separate it denotes expansion.

The apex impulse usually shows displacement, due either to pressure or hypertrophy, the former being more frequent.

Changes in the chest in the form of loss of respiratory movement and recession of the chest wall, the result of pressure on a large bronchus and consequent collapse of the lung, may occasionally be observed.

Palpation not only confirms the above, but is a more efficient means for their recognition. It often reveals the pulsation of tumors not detectable by inspection. When the tumor is in evidence, especially when it has perforated the anterior wall of the thorax, a heaving movement is felt peculiar in being expansile, and in this respect differing from the rising of a tumor over an artery. The amount of resistance in the sac walls depends upon the presence of clots or laminæ within the sac, being firm, if they are numerous, and yielding, if they are few. The sensation which an aneurysmal tumor imparts to the hand is that of a diastolic shock, frequently of marked intensity. This shock is an important sign of aneurysm and is due to the falling back against the aortic valves of a large quantity of blood, namely, that contained within the sac in addition to the normal flow. Frequently a systolic thrill and a purring fremitus are sometimes felt. When aneurysm is deeply seated bimanual palpation should be employed, placing the palm of one hand over the spine and that of the other over the area of the tumor anteriorly.

Percussion necessarily varies according to the distance of the tumor from the surface. When small and deep seated it is negative, but when large and near the surface there is always abnormal dulness. Aneurysm of the ascending portion of the arch on account of its forward and to the right growth is attended with increase of dulness on the right side of the sternum; those in the transverse portion give dulness in the median line extending toward the left, while those in the descending portion produce dulness in the left interscapular and scapular spaces.

In some cases compression of the lung produces dulness beyond the area of the aneurysm.

Auscultation gives varying results. Adventitious sounds may not be detected even in large aneurysms or they may be very decided. This variation is doubtless due to the depth at which the aneurysm is seated and the extent to which its walls are lined with laminæ, more especially the latter. A murmur or bruit, however, is usually audible. It is heard with the greatest intensity over the body of the tumor if present, and is transmitted to the vessels of the neck and along the course of the aorta. It is of a booming character and is usually systolic. A diastolic murmur may be added. The former probably arises in various ways, being the result either of vibration of currents passing into the sac or of eddies of blood within the sac which start from the current of blood as it enters the sac or from pressure of the tumor on the lumen of the aorta. In some instances there may be an associated obstruction of the aortic orifice causing a systolic murmur which is propagated by the sac. This is the source, according to some observers, of the systolic murmur heard in many cases of aneurysm. A diastolic murmur is often audible, due without doubt to a co-existing aortic insufficiency, the result of implication of the aortic valve. According to Gibson there are only four well-authenticated cases of sacculated aneurysm of the aorta in which there was a diastolic murmur with normal aortic valves.

An important sign, especially over a dull area, is the presence of a ringing accentuated aortic second sound which is generally heard in aneurysm of large size situated in the arch. (Osler.)

In some instances a systolic murmur is audible over the trachea. This is due to the transmission of the sound from the sac.

It is important to note that the signs of ausculation of aortic aneurysm are in many instances given very differently by different writers.

Auscultation of the heart very frequently shows the presence of aortic murmurs and it is important to observe that such adventitious sounds are more distinct over the aneurysm than in the aortic area.

Tracheal tugging, as a sign of aortic aneurysm, was first mentioned by Surgeon Major Oliver, who gave the following description of the method :*

"Place the patient in an erect position and direct him to close his mouth and elevate his chin to almost the full extent; then grasp the cricoid cartilage between the finger and thumb and use steady and gentle pressure on it. When dilatation or aneurysm exists the pulsations of the aorta will be distinctly felt transmitted through the trachea to the hand. The examination will increase laryngeal symptoms if any are present."

The value of this procedure as a means of diagnosis is generally accepted. It affords recognition of aneurysm in its earliest stages and when deeply seated.

X-RAY DIAGNOSIS, see Part VII.

Diagnosis.—As might be inferred from the symptomatology, the diagnosis of aneurysm of the aorta is sometimes difficult or impossible and sometimes, on the other hand, readily made. This variation depends upon the stage of development and the distance of the tumor from the surface. In the earlier stages, and when deep seated, the diagnosis is obscure. Pressure symptoms, such as have been enumerated, may be present to a greater or less degree, but are not pathognomonic since they occur in other conditions; they may, nevertheless, be regarded as decidedly suggestive.

The presence of a pulsatile tumor in which the pulsations are forcible, together with adventitious systolic murmurs and diastolic shock and ringing accentuation of the aortic second sound, is the most positive evidence of aneurysm of the aorta. In many instances, however, the diagnosis must be reached by exclusion. The presence of arterio-sclerosis and the history of the cause which led to it are important elements in diagnosis.

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* Lancet, London, 1891.

The conditions with which aortic aneurysm may be confounded, when a pulsating tumor presents itself, are tumor of the mediastinum, cancer of the stomach, abscess of the mediastinum, pulsating empyema, growths in the lungs, aneurysm of the innominate artery, and simple dynamic pulsation.

Tumors of the mediastinum in many respects closely resemble aortic aneurysm, and the differentiation is sometimes attended with great difficulty, as every pressure symptom may be present in both. There may be a pulsating tumor with an area of dulness resembling in area and extent that of aneurysm; there may be displacement of the thoracic viscera and even a systolic murmur. The means of recognition rest mainly upon the condition of the walls of the arteries, whether they are intact or not, the character of the aortic sound, the results of tracheal tugging, and the presence of a ringing aortic second sound, which is rarely if ever heard over a solid tumor. Moreover, pain is more prominent in tumor and pressure symptoms less so. The general condition of the patient is also a factor, being, as a rule, better in aneurysm than in tumor. In the latter there may also be implication of the cervical and axillary glands. The history of the case, as of syphilis, hard labor, etc., will assist. At the best, however, when aneurysm is deep-seated diagnosis is sometimes impossible.

Cancer of the stomach may occasionally press upon the thoracic aorta and suggest aneurysm. Errors may be avoided by noting that in aneurysm there is no cachexia; test meals show the presence of hydrochloric acid in the gastric juice, and gastric symptoms are mild or absent. Moreover, the physical signs of aneurysm, if present, will remove all element of doubt.

Abscess of the mediastinum is much less likely to be confused with aneurysm from the fact that it is attended with the general symptoms of suppuration and sometimes those of septicæmia, while direct pressure symptoms are not so marked as in the case of aneurysm. Here, also, the history of the case and physical signs lend valuable aid in avoiding error.

Pulsating empyema, if attended with a projecting tumor, may early prove deceptive and be mistaken for aneurysm. The points of difference are chiefly the absence of the firm, heaving pulsation and of diastolic shock, the presence of flatness posteriorly, the absence of pressure symptoms and alterations in the pulse, the more diffuse character of the pulsation, the extension of the area of dulness beyond the aortic area and the history of the case. Doubt may be removed by puncture with a hypodermic needle.

In very rare instances, growths in the pulmonary tissues of a syphilitic, tuberculous or cancerous nature may press upon the heart or aorta and propagate an impulse, and in some degree simulate aneurysm. In such cases confusion can usually be eliminated by the history and physical examination, which will usually show that the position of the tumefaction is not possible in aneurysm.

Aneurysm of the innominate artery is very difficult to distinguish from that of the aorta. The main features of difference are that the direction of the tumor in the case of the innominate artery is toward the right sternoclavicular articulation, the radial pulse of the right side is retarded, feeble or absent, and pressure is chiefly upon the right bronchial plexus, the right bronchus and superior vena cava. Sometimes, however, differentiation is impossible.

Simple dynamic or neurotic pulsation, without pain or pressure symptoms, is spoken of by Murray, Bramwell, Goodno and others. This phenomenon is observed in the episternal notch. The attending neurotic symptoms will prevent error in diagnosis.

Violent throbbing of the arch of the aorta in aortic insufficiency may be mistaken for aneurysm.

Prognosis.—The possible existence of aneurysm of the aorta without symptoms and the ever-present danger of rupture of its walls render the prognosis uncertain in the extreme. Spontaneous cures, however, not infrequently happen in small, sacculated varieties situated in the ascending portion of the arch. In these cases the cavity gradually becomes filled with successive layers of laminæ until it is entirely obliterated, when the walls shrink.

While it is not possible to forecast the future, there are certain factors which may be regarded as exercising a determining influence on the course of the disease. These are the condition of the arterial walls, the aortic valves, and the heart-muscle. The general condition, the surroundings and habits of the patient are also important considerations.

Generally speaking, the more positive the signs and the larger the aneurysm the more serious the condition. Notwithstanding its gravity, patients with aneurysm, even of very large size, have lived for many years; some as long as eleven years.

Aneurysm may terminate by rupture of the sac and hæmorrhage into the cavities of adjacent organs, or it may rupture externally or death may result from the effects of direct pressure, atheroma or secondary inflammation of neighboring structures, such as pericarditis, pneumonia, or other lesions of the lungs. If hæmorrhage occurs into the pericardium death occurs immediately from compression of the heart; if into the lungs it may either be immediately fatal or there may be repeated losses of blood; if into the pulmonary artery, vena cava or right heart, grave circulatory phenomena arise with the development of a systolic murmur, dropsy and heart weakness.

Treatment.—The treatment of aneurysm of the aorta in a large number of cases promises nothing and at the best is only palliative, yet a certain proportion of cases, if seen early, are amenable to its influence. Cures occur spontaneously, as shown by the presence at autopsy of shrunken aneurysmal sacs, and the various methods of treatment, however unsatisfactory, have a small proportion of successes to their credit. The great aim is to induce coagulation of the blood in the sac and subsequent contraction and obliteration of the latter, which is favored by retarding its flow and by lowering its pressure within the sac.

Various methods have been suggested to accomplish this end. The most important is that of rest and modification of diet. Rest must be absolute, both mentally and physically. It is evident that such a course requires less work of the heart by causing it to beat many times less and throwing less pressure upon the weakened and dilated blood-vessels.

Tufnel's Treatment.—Valsalva first practiced bleeding and starvation, Bellingham strict diet without bleeding. Tufnel, of Dublin, instituted a systematized plan of rest and diet which has been very generally approved and has since been known by his name. It consists of absolute rest in the recumbent posture, with a restricted dry diet, as follows:

For breakfast, bread and butter, two ounces; milk, two ounces. For dinner, two or three ounces of meat and two ounces of milk or claret. For supper, two ounces of bread and two of milk.

In this way it is hoped to reduce the volume of the blood and render it more fibrinous and thus to favor coagulation. Balford added the administration of potassium iodide. This treatment should be persisted in for several months. But it is easy to see that few persons would be sufficiently heroic to submit to it for so long a period, and doubtless in many cases some addition to the diet could be allowed. The class of cases to which Tufnel's plan is adapted is sacculated aneurysms in the early stages and those with large sacs communicating with the aorta by means of a small opening. Not a few instances of success by the method have been reported which seem to promise the most in the class of cases mentioned.

The following drugs are used with the idea of acting upon the sac:

Potassium iodide appears to be the only one which seems to have any real effect. The action of this remedy is not yet understood. It is believed to be due to its influence in reducing blood-pressure and diminishing the frequency of the heart-beat. Some observers attribute to it a specific action on the diseased vessel. When aneurysm is of syphilitic origin, as it is in many instances, this would certainly seem to be the case. However this may be, the administration of the remedy is frequently followed by beneficial results. One of its effects is the disappearance of painful sensations and arrest of anginal attacks. This is one of its most important actions. The remedy should be continued for months in doses of fifteen to twenty grains or more three times a day, with occasional remissions, especially if symptoms of iodism appear.

Ergotine has also been employed, but is not generally commended.

Astringents, such as acetate of lead, have likewise been used internally and appear to have had some effect, but the value of remedies of this class is negative.

Local Treatment.—While it seems to be quite generally agreed that rest, modified diet and potassium iodide are, from a medical standpoint, the best, if not the only means at command which promise anything in the treatment of aneurysm of the aorta, the surgical aspect of the question is different, for there is probably no subject in surgery in reference to which there is such a wide diversity of opinion. From time to time various methods have been brought forward and heralded as successful, only to be shown by subsequent experience to be unworthy of the claims made for them. The following is an enumeration of the most important surgical procedures which at various times have attracted attention.

Ergotine, subcutaneously, in the neighborhood of the sac was practiced by Langenbeck and others and successful cases reported. From two to four grains dissolved in glycerin or water, or seven to ten drops of Squibb's fluid extract are injected. Injections should not be made more frequently under favorable conditions than once in two or three days. Sloughing, cellulitis and oozing of blood from the aneurysm not infrequently follow this method, which has not been supported by experience and in consequence has become obsolete.

Injections in the sac of astringents, such as tincture of iron, acetate of lead, etc., are only mentioned to be condemned as worse than useless.

Galvano-puncture has been successful in some cases. Ciniselli reported twenty-three cases with four cures. In small sacculated aneurysm it is a hopeful resource. The method is as follows: First introduce into the sac a needle insulated except at its point; to this the positive pole is attached and the negative to a large electrode over the abdomen. The current is then allowed to pass from three to four hours. Before operating, the action of the apparatus should be tested in fresh blood or egg albumin. The danger from embolism is slight.

Another method is that of Loreta. Two needles are passed into the sac and a mild current of electricity is allowed to flow through, producing both an electrical and a mechanical effect.

Injections of gelatin were first introduced by Lancereaux a few years ago and successful cases reported. Subsequent experience, however, especially in this country,* has not substantiated these claims. Injections of a one per cent. solution of gelatin in normal saline are made into the sac. The strictest antisepsis is necessary, as suppuration is very liable to occur. Pyrexia is common. In a case reported by Z. A. Presman† (*Medicinskoie Obosrenie*, August, 1901) of aneurysm of the arch of the aorta in a woman forty-nine years old the treatment was successful. From August 21st to October 26th ten injections were given. After the eighth injection a general reaction characterized by fever developed. The injections were painful. Some writers speak of intense pain which lasted as long as six hours.‡

McEwen's method is as follows: Render the skin aseptic and insert a needle into the sac until it comes in contact with the wall on the other side. The needle may be left *in situ* for twenty-four to thirty-six hours. Another plan of McEwen is to introduce the needle as above described, then to move it about so that the opposite wall of the sac is irritated. The needle is then moved for about ten minutes; then another area is irritated without withdrawing the needle. In this irritation process the needle is only left in for a few hours. Success has followed this procedure in some cases.

The introduction of foreign substances was suggested and practiced by the late C. H. Moore, in 1864, in aortic and other inoperable aneurysms. In a case of aortic aneurysm Moore introduced twenty-six yards of fine iron wire. The result was the formation of a coagulum, but the patient died from sepsis. Since then various substances have been used for the same purpose, such as horse hair, fishing gut, watch spring, steel wire, copper wire, etc. The wire is introduced into the sac through a canula, being passed in obliquely. Considerable hæmorrhage may occur during the process. The success of this method has been so meager as not to warrant its use. The coagulum which results does not, as in nature, form on the walls of the sac, but on the foreign substance and embolism is very liable to result.

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* Guy L. Hunner, Bulletin, Johns Hopkins University, November, 1900.

† Philadelphia Medical Journal, March 15, 1902.

‡ Treat's International Medical Annual, 1901.

Distal ligation of arteries has been practiced for aneurysm of the innominate, the aorta adjacent to it and the transverse portion of the arch. The method of procedure is ligature of the right common carotid, the right subclavian or the left common carotid. Ligation of the left common carotid seems a less formidable operation and one that has been attended with more favorable results, which at the best scarcely justifies the operation.

Venesection has long been practiced. As a method of treatment it is not to be commended, but in certain conditions of dyspnœa and intense venous engorgement arising from mechanical obstruction from pressure of aneurysmal tumors it is said to have been employed to advantage.

TREATMENT OF SYMPTOMS.—From the character of the affection it naturally follows that palliation is a very important part in the treatment of aortic aneurysm. In the numerous instances, where from the nature and conditions of the case no plan of curative treatment is possible, the patient should lead as quiet a life as his circumstances and surroundings permit. All exertion should be avoided and intervals of rest in the recumbent posture, if possible, practiced. For palpitation, pain and præcordial distress the ice-bag may be applied. When there is marked evidence of arterio-sclerosis glonoine, four or five times a day, often proves useful. For violent throbbing, aconite in drop doses of the tincture is recommended. Other remedies may be prescribed according to indications, frequently with relief. Phenacetine and some of the other coal-tar derivatives sometimes palliate the neuralgic pain. Morphia, hypodermically, will be necessary in the latter stages, for the relief of pain and dyspnœa. Chloroform inhalations may alone be administered in advanced conditions of dyspnœa and pain not otherwise relieved.

When dyspnœa arises from pressure on the trachea or is due to bilateral paralysis of the vocal cords, tracheotomy may be necessary.

In all instances, whether or not any special line of treatment is adopted, the bowels should be kept free and straining at stool avoided.

PART III.

DISEASES OF THE BRONCHI, LUNGS AND PLEURA.

SECTION I.

DISEASES OF THE BRONCHI.

BRONCHITIS.

SYNONYMS.—*Trachelo-Bronchitis, Catarrhal Bronchitis.*

BRONCHITIS is a catarrhal inflammation of the bronchial tubes and trachea. It occurs as simple acute bronchitis, chronic and fibrinous bronchitis. Involvement of the fine bronchioles, called capillary bronchitis, is almost invariably accompanied by implication of the alveolar structure, and is now regarded as practically broncho-pneumonia, consequently, it is not discussed as a distinct disease process.

Simple Acute Bronchitis.

Simple acute bronchitis is inflammation confined to the trachea and large bronchial tubes.

Ætiology.—Predisposing influences are age, namely, infancy and old age, diathesis as evidenced by the susceptibility of persons with weakened constitutions, pulmonary, valvular or renal disease, and defective hygiene, such as living in badly ventilated, overheated apartments. The most frequent cause is chilling of the surface of some portion of the body or, as it is commonly called, "catching cold." Hence meteorological influences, as manifested by sudden changes of temperature, dampness, exposure to draughts of cold air, are among the most common causes.

It has been suggested that bronchitis is of microbic origin. While this is doubtful in ordinary forms, in epidemic varieties where it constitutes an important symptom, as in la grippe, it holds true, although the specific microbe has not yet been satisfactorily demonstrated.

Acute bronchitis arises secondarily in connection with a large number of diseases, especially the acute infections. The most notable of these is measles, where it forms a constant symptom, and whooping-cough. It may also be observed, although much less frequently, in typhoid fever, smallpox, diphtheria, etc. Gout, rheumatism and Bright's disease, by the retention of excrementitious matter in the blood, may produce acute bronchitis. Organic diseases of the nervous-system, such as locomotor ataxia, disseminated sclerosis and progressive muscular atrophy, are frequently attended with acute bronchial catarrhal conditions, which in many instances probably arise from the lowered state of the system, rather than from the direct action of these affections. The inhalation of irritant chemicals, such as chlorine, bromine and powder or dust of steel filings, coal or cotton, may induce the affection in some instances.

Morbid Anatomy.—The morbid changes are the same as those which occur in inflammations of the mucous membrane elsewhere, and are usually bilateral and confined to the trachea and larger bronchi. In many instances the process is the result of extension of an acute catarrh of the upper respiratory tract. In the early stages there is a hyperæmia, which is more marked around the secretory glands, and which varies in intensity according to the intensity of the inflammation. The mucosa shows reddened, arborescent areas which in the severe forms are more evenly and generally diffused, with decided swelling, the membrane presenting a bright red, velvety appearance. This change is due to œdema of the papillæ, the development of granulations and the obliteration of the longitudinal rugæ. Marked cellular infiltration of the mucosa occurs in ordinary cases on account of the tendinous character of the basement membrane, which is situated between the blood-vessels and the mucous membrane itself.

In the onset of these changes there is dryness of the membrane. This condition is quickly followed by hypersecretion, when the mucosa appears more or less covered with mucus.

Symptoms.—Simple acute bronchitis usually manifests itself as a common cold with coryza, sore throat, hoarseness, soreness and rawness of the chest, aching of the limbs, headache, pleuritic-like pains and some fever. There may or may not be a slight chilliness and sometimes a decided chill. The bronchial symptoms generally arise from an extension of the catarrhal process in the upper respiratory tract downward, and are those of a sense of tightness and irritation beneath the sternum with dry cough followed, as the secretion increases, by mucous expectoration.

The symptoms may appear in a more or less modified form or may be severe, hence the affection may be regarded as presenting two types, the mild and the severe. In the former the temperature may range for only twenty-four or forty-eight hours slightly above normal and the symptoms of aching and soreness may disappear after a few days, leaving behind a slight bronchial catarrh and cough. In severe forms there may be a decided chill and the symptoms of thoracic pain and soreness, aching of the back and limbs, general malaise, are more pronounced. The temperature ranges more persistently higher and registers from 100° to 102.5 or 103° F. There may be some oppression, but it does not amount to dyspnœa. The evidences of bronchial catarrh are much more marked and may be present for a fortnight or more. The cough may be very annoying, being frequent and harassing, in some instances causing sleeplessness, vomiting and involuntary urination.

In both the mild and severe forms of bronchitis as the expectoration becomes loosened and increased in quantity the cough becomes less troublesome.

A special form called gouty bronchitis is that which may occur in gouty subjects, sometimes as a precursor of gout, at others as a concomitant symptom, and at others again as a phenomenon resulting from retrocession of gout. The symptoms are those of bronchitis in its severe form, with pulmonary congestion and irregularity of the heart action.

Complications may occur in the form of capillary bronchitis or lobular pneumonia and atelectasis pulmonum, especially in infancy and old age. The imperfect expectoration at these periods of life also tends to cause the secretions to accumulate in the dependent portions of the lung and produce bronchiectasis. In measles and whooping-cough the tendency to implication of the finer tubes is always pronounced. The fine vessels are liable to become occluded with mucus and form areas of collapsed lung tissue and subsequently lead to broncho-pneumonia.

The course of the affection is from a few days to several weeks. In persons suffering from debility from any cause or with diathetic tendencies, it is liable to be protracted or to assume a chronic form.

PHYSICAL SIGNS.—In the mild form there may be almost complete absence of physical signs, or at the most a few loud sonorous or sibilant râles. *Inspection* in the severer forms may show some acceleration of the rate of respiration. *Palpation* shows transitory rhonchial fremitus if there is much narrowing of the calibre of the bronchi. *Percussion* reveals no change. *Auscultation* furnished the only positive evidence. In the early stages there are dry râles, which may be heard anywhere and which are transitory in character. There may be in addition some harshness of the respiratory murmur. As secretion increases the râles become more moist and bubbling. They will now be coarser or finer, according to the size of the tubes in which they are situated, and will change from time to time.

Diagnosis.—The recognition of bronchitis is a simple matter. The general symptoms, together with the presence of bronchial râles and absence of dulness on percussion and other physical signs, are conclusive. In some instances when the large tubes alone are involved the bronchial râles may not be detected, or coughing may cause their temporary disappearance.

It is scarcely possible to confuse simple bronchitis with any other disorder; in the severe form, however, care must be taken not to overlook the presence of a more serious condition. This caution is necessary, especially in regard to capillary involvement and broncho-pneumonia. In simple bronchitis the subcrepitant râles and areas of dulness on percussion which characterize broncho-pneumonia are absent, the respiration is freer, there is no real dyspnœa and the general symptoms are milder. Bronchitis may sometimes be confused with asthma and pulmonary tuberculosis in its earlier stages. It is distinguished from the former by the absence of the characteristic labored breathing, the loud rhonchi, the rhonchial fremitus and history of the case. Its differentiation from pulmonary tuberculosis is discussed under that subject.

Prognosis.—The prognosis is good. Only in the severer forms is there any gravity, and this is generally confined to young children, those advanced in years and those suffering from debility and diathetic tendencies. Under these conditions there is danger that the finer capillaries may become involved and broncho-pneumonia develop.

Treatment.—There seems to be an impression among the laity that a full dose of quinine or a Turkish bath are excellent to abort a cold. Whether the quinine has any real effect it is difficult to say, but it is certain that the bath is a doubtful expedient and not to be recommended, as the temporary relief obtained is more than counterbalanced by the subsequent exposure. In mild cases domestic remedies in the form of hot drinks, hot foot baths and sinapisms to the chest answer very well.

When the symptoms of bronchial inflammation are decided the patient should remain in bed until all symptoms have disappeared and convalescence is established. The temperature of the apartment should be maintained at from 68° to 70° F. The diet should be light and simple and the bowels kept free. In feeble persons and in conditions of debility the strength should be kept up by concentrated liquid nourishment and stimulants. Stimulants should also be given when the expectoration is tenacious and its expulsion attended with exhaustion. In severe cases the atmosphere of the sick room may be kept impregnated with steam. It is advisable for the patient to change his position from time to time, especially to avoid lying continuously on the back. This is to lessen the tendency to gravitation of the bronchial secretion to the base of the lungs.

Medicinal treatment varies according to the stage of the disease. In the first stage, that of arrested secretion, which is very short, the indications are to allay irritation and promote secretion. When the latter is affected the former usually follows. In the second stage, that of secretion, the indications are the removal of the same, and in severe cases support of the patient's strength. In the first instance the general condition is best met by the exhibition of aconite or bryonia.

The following are the chief remedies for acute bronchitis with these principle indications:

Aconitum, when there is fever and restlessness in the early stages. Fractional doses of the tincture half hourly to induce perspiration are beneficial.

Bryonia, for the sensation of rawness under the sternum, dry, racking or violent cough with turgescence of the face, sharp pleuritic pains, sense of aching and soreness throughout the body.

Antimonium tartaratum, one of the best remedies in both stages, either alone or in alternation with bryonia as mentioned. In the early stage it hastens secretion, in the later it facilitates expectoration by its "loosening" properties. In the later stage its special indications are the presence of loose, lumpy mucus, difficult of expectoration, much wheezing and rattling of mucus and tendency to implication of the finer bronchial tubes, especially in children and in aged persons.

Kali bichromas, when the mucus is stringy, tenacious, yellowish in color, tongue thickly coated, irritation of larynx, trachea and pharynx. The remedy may be used by inhalation in the proportion of two grains of the powdered drug in four ounces of water.

Ipecacuanha is useful, especially in children, being indicated when their breathing is asthmatic in character, with rattling of mucus in the bronchi, nausea, wheezing respiration with gastric and intestinal disturbances. In severe cases, when the secretion is excessive and difficult to expectorate, it affords relief.

Heroin or *codeine*, preferably the former, will be found excellent in palliating the cough. An eighth or sixteenth of a grain of heroin in simple elixir occasionally during the day proves very effective when the cough is annoying.

Hyoscyamus and *rumex* are both useful for irritating cough, the former when cough is worse at night and on lying down, with extreme sensibility of the trachea and larynx, the latter with similar indications in addition to excessive sensibility to air striking the neck and to any pressure over the trachea.

Scilla maritima, when the cough is spasmodic, with scanty expectoration and thoracic pain. This remedy also enters into the combination of old time cough mixtures for administration in the second stage.

Antimonii arsenis may be given in place of the tartrate in severe forms when in addition to the general indications for that remedy there is oppression, cyanosis with cold perspiration, anxiety, restlessness and debility.

Antimonii iodidum, while efficacious in simple stages, is especially useful when an acute attack of bronchitis supervenes upon an existing phthisical condition, where there is heavy yellowish muco-purulent expectoration.

Hepar sulphuris, when there are long paroxysms of loose cough with purulent expectoration in protracted cases and in those with laryngeal complication.

Ammonii carbonas, in three- to five-grain doses, will prove of service to facilitate expectoration when it is tenacious and its expulsion is attended with exhaustion, especially in feeble and aged patients.

Senega may be used under the same conditions as the ammonium carbonate.

In gouty bronchitis the treatment should be directed to the gout, colchicum, soda salicylate and alkalies being the chief remedies.

Chronic Bronchitis.

SYNONYMS.—*Chronic Bronchial Catarrh, Chronic Trachelo-Bronchitis.*

Chronic bronchitis is a chronic catarrhal inflammation of the mucous membrane of the trachea and bronchi. It arises primarily and secondarily and occurs as simple chronic, dry and putrid bronchitis and bronchorrhœa.

Ætiology.—The affection is more prevalent in cold, damp and changeable climates and in winter. In many cases it disappears in summer and reappears in winter. It is also more frequent among men than women from the fact that the former are more exposed to its causal influences. Age also seems to be a factor, as chronic bronchitis is commonly observed in those of advancing years. Sometimes, however, it occurs in the young. Repeated attacks of acute bronchitis may result in the primary or simple chronic form, hence, exposure, climatic influences and inhalation of irritants may be the initial causes. In association with these factors there is frequently some predisposition, such as age or diathesis. Simple chronic bronchitis may also be traced to acute bronchial catarrhs which arise secondarily in connection with some other affection, such as measles, whooping-cough or broncho-pneumonia, but which fail to resolve after subsidence of the acute lesion and assume a chronic course. It is this type which is liable to affect young persons.

Osler speaks of a form of chronic bronchitis occurring in women between twenty and thirty years of age. The cough is described as more pronounced in the morning and accompanied by a profuse muco-purulent expectoration. The physical signs are negative. The ætiology is obscure, but a gouty and tuberculous tendency were observed. The affection does not appear to affect the general health and may continue indefinitely.

Secondary chronic bronchitis, the usual form of the affection, depends upon a variety of ætiological conditions which may be summarized as follows: diatheses, such as gout, rheumatism, syphilis, Bright's disease, chronic alcoholism, pulmonary tuberculosis, emphysema, rachitis, scrofula and valvular lesions of the heart. In these conditions it is the result of the altered state of the blood from retention of toxic matters. In chronic alcoholism, in addition to the retention of toxines in the circulation, alcohol acts by producing irritation and hyperæmia of the mucous membrane. In pulmonary tuberculosis it forms part of the process which arises from the presence of the bacillus, at the same time it may be present before the inception of the bacillus, being dependent upon other causes and in itself a factor leading to the development of tuberculosis.

In emphysema and cardiac diseases chronic bronchitis appears secondarily as a product of interference with the circulation of the lungs which results in hyperæmia, being more frequently found in mitral disease.

Morbid Anatomy.—The changes vary according to the diseases with which the bronchitis is associated. At first only the mucous membrane is affected, but in time all the bronchial structures and the peri-bronchial tissue become involved. The mucosa undergoes a variety of changes, in some places becoming, it may be, very much thinned, so that longitudinal elastic fibres stand out prominently, presenting a lattice-like appearance, in others it may be granular or thickened from cellular infiltration. There also may be ulceration at certain points, more especially of the mucous follicles. The follicles may likewise present some hypertrophy, in some instances undergoing atrophy, in others hypertrophy. Sometimes there are areas from which the epithelia have been denuded. The membrane is of a dull, grayish hue. These changes are attended with weakening of the walls of the tubes which at certain points, in cases of long standing, may undergo dilatation, either sacculated, cylindrical or fusiform. In some instances the dilatation may be so excessive as to suggest on examination the presence of a cavity. This occurs more especially in the centre of the lung. There is always a tendency on the part of the secretion to collect in these dilatations.

The air vesicles may likewise be affected and dilate, and in consequence emphysema is a frequent accompaniment. There is often a dilatation of the right side of the heart.

The character of the secretion is usually muco-purulent, but it varies. Its color may be yellowish from the presence of pus cells, or dark gray from the admixture of degenerated epithelium or decomposed blood. Sometimes it may be a jelly-like mass of muco-pus; occasionally, it may be thin and watery from excess of serum.

Symptoms.—Variability arising from the presence of associated lesions and dyscrasias characterizes the clinical course of chronic bronchitis. The chief symptoms are cough, shortness of breath and expectoration. In the earlier periods the general health may remain unaffected, but later evidences of emphysema, cardiac dilatation and visceral disturbances become manifest.

The cough is inconstant and appears in different forms, depending upon the amount of secretion, the weather and season. During warm weather it may be absent, returning each winter and persisting until the following summer. It is commonly aggravated in the morning owing to the bronchial secretion accumulating during the night. It may also be very troublesome during the night. In some patients again it may appear in recurrent attacks.

The character of the sputum differs according to the type of bronchial catarrh. It may be copious, muco-purulent, watery, scanty or putrid. Microscopically, it consists of broken-down epithelia and pus cells, blood-corpuscles, granular detritus, micro-organisms and shreds of tissue from the bronchi. It is chiefly in regard to the variability of the sputum that the different clinical forms of chronic bronchitis are recognized.

Dyspnœa is common to all forms of chronic bronchitis; it is usually, however, only apparent on exertion, disappearing immediately when at rest. It is due more to the associated emphysema and cardiac dilatation than to changes in the bronchial mucous membrane. In some instances there may be true bronchial asthma. Fever is rarely present. Evidences of diathetic tendencies may occasionally be observed. Sometimes patients suffer from gouty symptoms and chronic bronchitis and manifestations of gout, for example eczema, alternate with bronchitis. In other cases both may be present at the same time.

BRONCHORRHŒA.—The distinguishing symptom in this variety is the excessive amount of secretion which, in exceptional cases, may amount to three or four pints in twenty-four hours. The sputum may be very liquid and watery, but more often it is thin, with masses of greenish or yellowish muco-pus floating in it. When allowed to settle in a glass a layer of pus falls to the bottom, above which there is a layer of serum covered with froth.

Bronchorrhœa is commonly observed in elderly people, although it may occur in the young. It may exist a long time without apparent damage to the lung or detriment to the general health. On the other hand, it may lead to dilatation of the bronchi and ultimately to fetid bronchitis.

FETID BRONCHITIS.—In this form the sputum is extremely offensive, and the foul odor may permeate the whole apartment. Putrid expectoration occurs with bronchiectasis, gangrene and abscess of the lung, decomposition within phthisical cavities and empyema with perforation of the lung, and in rare instances independently of these lesions. The sputum is usually abundant, thin, grayish-white, separating into layers on standing. The lower layer consists of thick sediment, in which are sometimes found yellowish masses the size of peas, called Dittrich's plugs.

Occurring with the above-mentioned conditions, the symptoms obviously present considerable variation, the most constant necessarily being those of sepsis.

In severe forms there is danger of pneumonia and pulmonary abscess. Metastatic abscess of the brain has been found in association with fetid bronchitis.

DRY BRONCHITIS.—This form, described by Laennec (*catarrhe sec*), is not common. It is characterized by paroxysms of severe coughing attended with little or no expectoration. If sputum is raised, it is frequently tenacious and difficult of expulsion. In many instances the coughing is sufficiently violent to cause vomiting. Elderly persons with gout or emphysema are most subject to the affection.

Complications.—While chronic bronchitis in itself does not directly endanger the patient's life, the sequelæ may be very grave. Emphysema most always develops. Under the influence of recurrent bronchial attacks and violent paroxysms of coughing it is usually progressive and ultimately leads to dilatation of the heart. It is also especially conducive to asthma. The state of the bronchial tubes and mucous membrane is furthermore favorable to the development of capillary bronchitis, atelectasis, fibroid induration of the lung, and furnishes a nidus for the propagation of the tubercle bacilli.

Congestion of the abdominal organs, chronic interstitial nephritis are also not infrequent results of the sequence of changes which follow the disturbance of the pulmonary circulation.

PHYSICIAL SIGNS.—*Inspection* is negative in simple chronic bronchitis. In those cases associated with emphysema the characteristic signs of this lesion vary in extent, according to the amount of lung involved in the emphysematous condition. *Palpation* may reveal rhonchial fremitus. *Percussion* is usually normal, but may, according to some observers, disclose dulness over a limited area due to the accumulation of secretion in a dilated bronchus, on the removal of which the dulness disappears and sometimes gives place to tympanitic resonance. The presence of emphysema may likewise give diminution of the normal fremitus and a higher resonance to percussion. Vesico-tympanitic and even tympanitic resonance may be present from relaxation of the lung tissues, especially in the lower portion of the lung. *Auscultation* may show no recognizable change. On the other hand, it may disclose areas of weakened respiratory murmur from the influence of the secretion or from the more direct effects of inflammatory changes. There may also be harsh respiration, sometimes feebleness and harshness may alternate. Râles of all varieties occur, sonorous, sibilant with moist râles of all sizes. When widespread they point to asthmatic tendencies. The râles are heard to the best advantage posteriorly and are further distinguished by not being localized. The presence of gurgling râles when apparently confined to a given area is suggestive of dilatation of a bronchial tube.

Diagnosis.—The recognition of chronic bronchitis is usually unattended with difficulty. If secondary, a knowledge of the primary lesion is important, therefore the heart and urine should be carefully examined.

In reference to the differential diagnosis and the presence of complications the observer will be called upon to exercise some care. One of the foremost questions is to determine whether or not pulmonary tuberculosis is present. This is difficult when the latter is in the incipient stage. The following are points of differentiation: The clinical history; in bronchitis there is no fever, no great loss of flesh and the general health is not impaired. In tuberculosis there is localized consolidation usually at the apex early, while in chronic bronchitis the vesicular murmur remains. The recognition of the bacilli in the sputum is obviously decisive.

Fetid bronchitis occurring primarily may be differentiated from abscess by the presence in the sputum of the latter of shreds of lung tissue, including elastic fibres, crystals of cholesterin, hæmatoidin and blood pigment. Also by localized dulness and broncho-cavernous respiration. In gangrene the elastic fibre is absent on account of the presence of a ferment which causes its solution.

Chronic bronchitis may be distinguished from bronchiectasis by the general difference of the physical signs which in the latter are unilateral and confined to limited areas.

Prognosis.—The course of the affection is extremely protracted and in the uncomplicated forms the patient may live many years. Recovery is exceptional, although improvement is often observed. The prognosis may be said to depend mainly upon the nature and extent of the associated lesion and sequelæ and complications.

The affection naturally tends to weaken the lungs and to put them in a condition receptive for disease, especially pneumonia and broncho-pneumonia, which are quite commonly the cause of death in aged persons. Implication of the heart and kidneys is necessarily unfavorable.

In all instances great care should be exercised before giving any decided opinion as to the future.

Treatment.—The most important element in the treatment of chronic bronchitis is climate. Removal to a warm, equable, dry climate should be enjoined in all instances whenever practicable. Residence in southern California, southern France or Florida in winter will prove of great benefit, and in the recurrent form, which appears only in cold weather, will act as a prophylactic. Those who are unable to leave their homes during winter should avoid as much as possible all exposure and should remain indoors during stormy or damp weather; at the same time they should take care that their apartments are free from vitiated air and that good ventilation is maintained. Woolen underwear should be worn by all bronchitic persons even in mild climates when light-weight garments should be substituted for the heavier of the northern winter. The general nutrition should be kept up by suitable diet as in other chronic affections.

Before determining on any line of treatment it is important to ascertain, if possible, the existence of any associated affection, diathetic or otherwise, and govern the selection of remedies accordingly, bearing in mind that gout and rheumatism are the most common, that pulmonary emphysema and cardiac dilatation are usually secondary, and that Bright's disease may play both a primary and a secondary rôle.

When in elderly patients the arteries are atheromatous, the urine slightly albuminous and the bronchial attacks associated with evidences of defective elimination and gout, the treatment should be directed toward the underlying causes. Under these conditions there are often cardiac complications and emphysema. In the case of the former digitalis, nitro-glycerin and allied remedies will aid in not only strengthening the heart muscle, but also relieving the mucous membrane of the lungs by lessening the pulmonary congestion and tendency to stasis.

The length of the list of remedies which have been recommended for chronic bronchitis is suggestive of the inefficacy of drugs in its treatment. While it is true that cure is seldom thereby effected, decided relief and valuable assistance may frequently be obtained. One reason which accounts for the variety of remedial agents is the diversified ætiology and frequency of associated lesions.

The following are some of the principal remedies with their general indications:

Ipecacuanha is useful in many forms of chronic bronchitis, especially when associated with asthmatic symptoms.

Terebinthina, in some of its forms, is often beneficial, especially terpin.

Creasotum, in all varieties of chronic bronchitis, especially the fetid.

Hepar sulphuris, when there is a loose cough with muco-purulent expectoration, aggravated in the early morning hours. There may be considerable rattling, but not much expectoration.

Kali bichromas, when the sputum is very tenacious and stringy, with wheezing respiration. The remedy may also be used by inhalation.

Kali carbonas, indicated by the presence of "stitches" in the side, asthmatic breathing, spasmodic cough, gagging and difficulty in expectorating, aggravation of condition in early morning.

Drosera, when the cough is hard and paroxysmal, sometimes exciting vomiting, both with and without emphysematous conditions.

Iodium is beneficial in many of its forms. It may be used with good effect when the cough is inclined to be dry, hacking and exhausting. General emaciation with night-sweats and disposition to phthisis are additional evidences.

Potassii iodidum acts with excellent effect in the course of chronic bronchitis, for which it is difficult to assign a definite cause, especially in those depending upon diatheses, particularly syphilis and gout. Scanty secretion is an indication.

Antimonii iodidum, for spasmodic cough, especially in the morning, with free muco-purulent expectoration, sometimes with a sweetish taste. There may also be loss of flesh and night-sweats.

Arsenici iodidum, beneficial in cases with muco-purulent expectoration, anæmia, tendencies to fibroid degeneration of the lungs or phthisis.

Arsenicum album, when there is dyspnœa from emphysema and cardiac disease, dry, wheezing cough, scanty expectoration, anæmia and loss of flesh.

Grindelia robusta, an excellent remedy when there are asthmatic symptoms, paroxysms of cough with little expectoration, yet râles are present; it lessens the cough.

Acidum benzoicum, while useful in facilitating expectoration in chronic bronchitis, acts best in gouty persons with offensive, high colored urine and other evidences of faulty metabolism.

Sulphur is often indicated in patients with gout or other diathetic tendencies, bronchorrhœa and associated skin lesions.

Segena, in bronchitis of elderly persons where there is copious tenacious sputum raised with difficulty, irritating and paroxysmal cough.

Antimonium tartaratum, while more especially a remedy for acute bronchitis, is also used in the chronic form, particularly when there is the acute inflammation engrafted upon the old lesion with emphysema, moist râles and defective aëration depending upon the lungs and not the heart.

Rumex crispus, for dry bronchitis with hyperæsthesia of the mucous membrane of the respiratory tract, cough, aggravated by eating, lying down, talking or contact with cold air.

Ammonii chloridum, when a stimulating effect is desired.

Ammonii carbonas, in old and feeble patients when the expectoration is raised with great difficulty or the patient makes little effort to expel it. Failure of the respiratory and circulatory functions with coarse râles and oppression; bronchiectasis, associated cardiac and renal disease are its principal indications.

Stannum, when the sputum is abundant, of a greenish color and sweetish taste, with weakness and sensation of emptiness of the chest and dilatation of the bronchi.

Stanni iodidum, when the expectoration is copious, free and densely purulent. It is suitable for simple forms of chronic bronchitis, as well as those with bronchiectasis and tuberculosis.

Lycopodium, useful in those forms associated with gout and phthisis when there is acid dyspepsia, excess of uric acid and constipation.

Calcarea, in the form of the carbonate, is valuable in chronic bronchial catarrh occurring in children with rachitic or tuberculous tendencies and feeble assimilation. Children who are fat, pale and flabby, with a disposition to profuse perspiration about the head, with enlarged glands, distended abdomen, light colored evacuations, and offensive diarrhœa are those in whom the remedy is especially indicated.

Anodyne remedies are sometimes necessary and of these heroin stands pre-eminently first, and codeine second; either may be given in the tablet form or in a simple elixir or in syrup of tolu. The following is recommended for the cough, to be taken in one dose: Sodium bicarbonate, grs. xv; sodium chloride, grs. v; chloroform spirits, ♏v; anise water and hot water, equal parts.

Alkaline mineral waters are often of decided benefit, especially those of Ems, Vals and Vichy. In lieu of the waters fifteen drops of liquor potassæ, U. S. P., may be taken in a glass of milk. Alkalies appear to favor the expectoration.

Inhalations are often beneficial, especially in the putrid form. They may be prepared by pouring the remedy in boiling water in an inhaler.

℞	Benzoini compositi,	ʒj.
	Aquæ bullientis,	Oj.

Inhale vapor night and morning.

℞	Acid carbolici,	gtt. iij–vj.
	Aq. bullientis,	Oss.

℞	Potas. bichromatis,	
	Aq. bullientis.	

℞	Acidi carbolici,	gtt. iv.
	Ol. eucalypti,	♏ij–iv.
	Aq. bullientis,	℥j.

For inhalation when expectoration is fetid.

℞	Terebini,	gtt. v–xl.
	Magnesiæ carbonatis,	gr. xx.

Sig.—Use a drachm to a pint of water at a temperature of 140° F., inhale for ten minutes night and morning.

℞	Ol. terebinthinæ,	♏v.
	Aq. bullientis,	Oj.

Temperature of water 155°.

℞	Creasoti,	♏v–vj.
	Magnesia carbonatis,	q. s.
	Aq. bullientis,	Oj.

Inhale when there is dilatation of the bronchi and retention of mucus.

℞	Iodii,	ʒj.
	Alcoholis,	℥ij–iv.

Mix in flask and add

	Terebinthinæ,	℥ij, ℈ij.
	Spiriti lavendulæ,	ʒx.

Inhale when expectoration is fetid and profuse.

For other forms of inhalation the reader is referred to Bronchiestasis and Pulmonary Tuberculosis.

FIBRINOUS BRONCHITIS.

SYNONYMS.—*Plastic Bronchitis, Croupous Bronchitis, Pseudo-Membranous Bronchitis.*

Fibrinous bronchitis is an inflammation of the bronchial mucous membrane characterized by the formation of a pseudo-membranous or croupous exudate, which on expulsion is found to be a mould of the bronchial tubes and its branches. The affection may be either acute or chronic, but is commonly the latter. In both instances it is very rare. Bronchial casts are also formed in diphtheria and in croupous laryngitis from extension of the exudation downward; casts of blood clots are likewise expelled in hæmoptysis, but these formations are entirely distinct from those under consideration.

The affection has long been known, having been referred to by Galen.

Ætiology.—The causation of the affection is unknown. It appears to be more common in males than in females, occurring twice as frequently, and between the ages of twenty and forty, although it may occur at any age. Spring seems to favor its development, as most of the cases are observed at that season. Occasionally, it has been encountered in an endemic form, being confined to groups of persons in certain localities. Its association with other affections is recognized, especially tuberculosis. It is also found with chronic bronchitis, chronic pleurisy with consolidation, emphysema, pneumonia, fibroid induration of the lung, cancer of the lung, stenosis of the bronchial tubes, cardiac disease and certain skin affections, such as herpes, pemphigus and impetigo. But whether these associations are real or accidental it is impossible to state, owing to the insufficient clinical data which necessarily follows from the rarity of the disease.

Hereditary influences are mentioned by some writers, but there does not appear to be sufficient evidence to substantiate these claims.

Morbid Anatomy.—The pathology of fibrinous bronchitis is as obscure as is its ætiology. As primarily expelled the exudate is a rounded mass mixed with blood and mucus. When cleansed and unrolled it is found to be an accurate dendritic cast of a portion of the bronchial tract. The termination of the infundibula may even be moulded. The false membrane is of a whitish or gray color, with more or less firm adhesion to the bronchial surface, and is detached by coughing. It is composed of true fibrillated fibrin, in which are embedded a number of leucocytes, cylindrical epithelia, ëosinophile cells, hæmotoidin crystals, occasionally fat globules, Curschman's spirals and Charcot-Leyden crystals. It is concentrically laminated in structure forming a tube except when from the small branches of the bronchi, when it is solid. The size of the casts varies from one-half to ten inches long, and is sometimes longer. They vary greatly in number and form in different portions of the bronchial tract and re-form at the same points after expulsion. The application of acetic acid causes them to swell.

The mucosa of the tubes from which they come may be either intensely congested or very pale. Microscopic examination discloses diminution of the epithelia with œdema and serous infiltration of the submucous tissue. As far as the bronchial inflammatory process is concerned, it may be either diffused or localized.

Symptoms.—In the acute forms the symptoms may be preceded by those of simple acute bronchitis or they may appear abruptly. The onset is generally characterized by a chill, followed by considerable fever and dyspnœa. The latter resembles asthmatic breathing and is more or less pronounced according to the amount of bronchial surface involved; when this is extensive the dyspnœa becomes very urgent and in extreme cases death may ensue from asphyxia. Cough, with very rare exceptions, is continued and harassing and often has a peculiar tone. At first it is accompanied by the expectoration observed in simple acute bronchitis, but later, frequently during a severe paroxysm, the membrane is expelled with mucus, muco-pus and blood. In some cases there may be decided hæmorrhage. Some emphysema is almost always present.

The expulsion of the membrane is followed by relief of the dyspnœa, subsidence of the fever and general amelioration of the symptoms. This improvement continues until the formation of another exudate, which may occur in a few hours or several days, when there is a recurrence of the symptoms.

Some writers state that it is possible for the cough to be absent and expectoration to be free from casts. Under these conditions the condition may fail to be detected until there is a post-mortem examination, that is, the casts remain *in situ*.

The course of acute fibrinous bronchitis is from five or ten days to three weeks. The attacks may appear only once or may be recurrent. Favorable signs are decrease of dyspnœa and diminution in the number of casts expelled. Increase of dyspnœa, on the other hand, especially with retraction of the chest walls and thoracic pains, appearing late in the course of the affection, are decidedly unfavorable.

Complications may arise in the form of broncho-pneumonia, as might be expected from collapse of areas in the lung.

In the chronic variety the initial symptoms are usually those of bronchitis. There is not necessarily any fever. The cough becomes more troublesome and paroxysmal. Dyspnœa and cyanosis may be pronounced. Examination of the expectoration shows rounded masses which when unrolled and cleaned are found to be true casts of the bronchi. They are the same in color and anatomical structure as in the acute form. Frequently, the sputum is blood-stained and sometimes the expulsion of the casts may be attended with hæmorrhage.

Chronic fibrinous bronchitis is less severe than the acute form. Its course is characterized by periods of remission, the intervals being a week or several years. It is said in some instances to have existed for fifteen years. In some cases regularity of reappearance has been observed. Its recurrence in the spring has been noted.

PHYSICAL SIGNS.—The physical signs are those of severe bronchitis in association with those of obstruction in the bronchi of the affected areas. At these points, as a result of diminution of the amount of air entering the lung, palpation will show decrease of tactile fremitus, while local expansion and the normal respiratory murmur are diminished or absent. Percussion over the unaffected portion of lung is clear or hyper-resonant unless there is atelectasis, when there is dulness. Over the involved area there is the normal resonance unless the area implicated is extensive, when there may be loss of resonance owing to excessive accumulation or actual consolidation complicating the situation. Removal of the casts is followed by return of the normal respiratory murmur in uncomplicated cases.

The signs of fibrinous bronchitis being dependent upon the presence of the casts, they naturally vary according to the discharge and recurrence of the same.

Diagnosis.—The diagnosis of fibrinous bronchitis depends upon the recognition of the casts. The fibrinous exudations, which are sometimes expelled as moulds in diphtheria and pseudo-membranous croup should be excluded. As a rule, the history of the case is sufficient to prevent any confusion, but should doubt arise, bacteriological and microscopical examinations of the exudate should be made.

Broncho-pneumonia or pneumonia may coëxist with fibrinous bronchitis, hence the signs of these affections may also be present.

Prognosis.—In the acute variety the outlook is very grave, as a large proportion of the recorded cases have been fatal. The nature of the complications, if such are present, necessarily greatly influences the course and termination, especially pneumonia, either bronchial or croupous, which must be looked upon as especially unfavorable. Atelectasis also lends additional gravity to the situation. The chronic form is attended with much less danger, and unless there are grave complications there is seldom a fatal termination.

Treatment.—While this should be conducted on the same lines as in simple bronchitis, the aim to loosen and facilitate the expulsion of the membrane is an additional factor of great importance. For this purpose various measures have been recommended, the most trustworthy being inhalations of the steam of lime water or of some other alkaline carbonate. Plain steam may also be used Kali bichromate, also the iodide, are also commendable for inhalation. Sprays of papoid and papayotin and kindred solvents have been used, it is claimed, with success. When there is danger of asphyxiation and immediate action is required, other remedies having failed, an emetic should be administered, but otherwise emesis should be deprecated as too depressing.

As far as internal remedies are concerned, kali bichromate, iodine, bromine, spongia, kali iodide, may be used according to special indications pointing to their selection. Pilocarpine, through its action of inducing bronchial secretion, has also been employed with good results. Ewarts recommends intralaryngeal injections of oil.

BRONCHIECTASIS.

SYNONYMS.—*Bronchiolectasis, Dilatation of the Bronchi.*

The derivation of the term bronchiectasis, first described by Laennec in 1819, from Βρόγχος, the bronchus, and ἔκτασις, dilatation, aptly define its significance. The process may be partial or universal, and except in very rare instances of congenital origin it is always secondary.

Ætiology.—Weakness and loss of elasticity of the bronchial walls, whereby these structures are rendered unable to resist inspiratory and expiratory strain, is unquestionably the chief factor in the production of bronchiectasis. The causes which may lead to the condition are found in inflammation of the bronchi and contraction of lung tissue. Frequently, both influences are operative.

The process may be either acute or chronic. In connection with the acute form, acute bronchitis and broncho-pneumonia are the chief causes, especially in children; in the chronic form chronic bronchitis is accountable for the greater proportion of cases. Next in order of frequency is phthisis.

Acute bronchiectasis or bronchiolectasis is a disease of children. It cannot be differentiated clinically in life from capillary bronchitis, or broncho-pneumonia with which it is associated. The chronic form is that which is usually meant when the term bronchiectasis is used. It may occur at any time of life and is more frequent in men than in women. Other causes are bronchial obstruction arising from compression by aneurysm, growths and glandular enlargements, hydatids, stenosis from syphilitic ulcerations and foreign bodies in the air-passages. Causes operative through contraction of the lung tissues and inflammatory processes conjointly are interstitial pneumonia, tuberculosis, chronic pleurisy with exaggerated thickening and adhesions, emphysema, empyema and malignant disease.

The inhalation of dust and foreign bodies in a state of minute division was regarded by Hamilton as a cause.

Universal bronchiectasis may arise from interstitial pneumonia, or it may be congenital.

Morbid Anatomy.—Two varieties of dilatation are generally recognized, the cylindrical and the sacculated. In rare instances a fusiform variety may also be observed.

In the cylindrical form, which is the most frequent, the bronchial tube may be dilated throughout its entire length from the root of the lung to the pleural surface. Its calibre may increase as it descends. Sometimes it is quite regular, at others trabeculæ and thickenings cause it to appear uneven. The latter condition is common in the cylindrical form. More frequently the medium size and smaller tubes are affected. Post-mortem recognition of bronchiectasis is usually unattended with difficulty. By cutting through the lung the surface thus disclosed shows many small, tubular orifices which may be readily opened. Small dilated tubes may be mistaken for normal ones of coarse calibre. This error may be avoided by comparing the lumena, which show irregularities in the case of dilatation.

In saccular bronchiectasis the dilatations are spherical or oval, emerging from the tube either gradually or suddenly. They are single or multiple and may attain a considerable size, even as much as two or three inches. The surrounding lung tissue is usually indurated and contracted. The bronchus leading to the cavity may be normal, dilated, stenosed or even obliterated.

In partial dilatation the cylindrical variety prevails, although the two are often present.

In diffuse or universal bronchiectasis, sacculated dilatation is the usual form. The lung is the seat of numerous small sacculi, the entire bronchial tree consisting of a series of the same opening one into the other. The dilatation may result in the formation of cysts beneath the pleura with cirrhosed lung tissue intervening. The surface of the bronchial mucous membrane may be smooth and glistening and possibly free from ulceration or erosion, except in the dependent portion of the lung. Frequently, both cylindrical and sacculated dilatations are found together. Sometimes a single bronchiectatic dilatation will be observed, especially in chronic bronchitis or emphysema. In acute bronchitis and broncho-pneumonia, particularly in children, cylindrical dilatation of a number of small and medium size tubes frequently occurs. In protracted cases of broncho-pneumonia and chronic bronchitis dilatations of this type may reach great size. In phthisis dilatations are commonly located at the apex; in chronic pleurisy and emphysema, at the base.

The walls of the bronchi at the seat of dilatation often show important changes. As the process advances the superficial layer of the mucous membrane may become vascular and thrown into folds, or it may be thin and dense. The epithelia display various changes, being thickened, attenuated or entirely absent. The muscular structures undergo stretching and lose their tone and elasticity, while the fibrous tissue, glands and cartilage atrophy and sometimes disappear. In cylindrical and sacculated bronchiectasis, particularly the latter, the mucous membrane may ulcerate. This is doubtless due to the presence of bacteria and to retention of secretions which become exceedingly foul. The ulceration may sometimes be the seat of a tuberculous process and extend into the parenchyma of the lung forming large cavities. Occasionally, it may lead to gangrene.

Symptoms.—Peculiar sputum and paroxysmal cough are the distinctive features of bronchiectasis. The symptoms of the affection upon which it is dependent will obviously be in evidence, and unless the dilatation be marked, may predominate to such a degree as to obscure its recognition. This is readily understood when the nature of the primary lesions is taken into consideration.

The sputum is the most characteristic symptom and alone is diagnostic. It is muco-purulent, of a dirty, greenish-yellow color and offensive, acrid odor. It may have all the horrible offensiveness of putrid bronchitis. Another feature is that it is raised up in mouthfuls. On standing in a glass it will be seen to separate into three layers, the lowest being that of a thick sediment composed of epithelia in various stages of degeneration and granular débris; the second, a thin sero-mucous fluid and the uppermost a brownish froth. On microscopic examination the sputum shows the presence of pus corpuscles, fatty and acid crystals in bundles, and, occasionally, Charcot-Leyden crystals, with sometimes hæmatoidin and red blood-corpuscles sufficient to color it, also various bacteria. There will be no elastic tissue present unless there is ulceration of the bronchial walls.

The cough commonly occurs in morning paroxysms, being apparently excited by change of position. It is possible that the dilated portion of the bronchi is thereby made to empty itself into normal tubes, and as soon as the sac becomes full and empties itself cough again is repeated. This continues until the sac is emptied, when there is an interval of rest which may last for hours. Respiration in milder degrees of bronchiectasis shows no change except on exertion, when there is dyspnœa. In more pronounced cases dyspnœa is constant. There is no fever; if any develops in the course of the affection it is from other causes. Clubbing of the finger nails is a prominent symptom.

Possible complications are both numerous and various. They are as follows: Febrile disturbances due to absorbtion of septic matter, metastatic abscesses, cerebral, pulmonary and peritoneal, especially the first; pneumonia, pleurisy, empyema, emphysema, pulmonary gangrene, pneumothorax from ulceration, pulmonary fibrosis and calcareous degeneration of the bronchial walls.

PHYSICAL SIGNS.—These are lacking in distinctive features and frequently are negative. When recognizable they are, generally speaking, posterior and towards the base of the line, and are those of a cavity including tympanitic percussion, bronchial and amphoric breathing, bronchophony or pectoriloquy, according to the size, situation and state of the cavity, whether full or empty, and the condition of the surrounding lung tissue.

Inspection shows only such changes as may attend the primary lesion, there being none which can be said to be characteristic of bronchial dilatation itself.

Palpation usually gives increase of fremitus over the bronchiectatic areas. *Percussion* in the earlier periods may disclose no change, while in the advanced it may show marked variations. If the dilatation is near the surface it gives higher resonance. If consolidation is excessive there is necessarily dulness which will disclose areas of clearness according to the location of the dilatations. The presence of emphysema necessarily influences these signs.

Auscultation shows signs which vary according to the character of the primary affection and location of the cavity. There are usually evidences of bronchitis and at various points cavernous respiration. If the cavity is filled with secretion the respiratory murmurs at the base of the affected lung are feeble or absent and vocal resonance diminished. When empty the murmur is broncho-cavernous or cavernous with or without pectoriloquy. If the cavity is near the surface there will be pectoriloquy, varying in intensity, according to the depth of its situation. A prominent feature is the presence of râles of large size. They may be of all kinds, sibilant, sonorous, squeaking and croaking. The latter term was suggested by Ewart as peculiarly fitting. Over these same areas where the râles are heard there may be harsh or cavernous resonance.

In those cases where the cavity is associated with secondary pulmonary consolidation there may be signs, in addition to those mentioned, of consolidation, either diffuse or general. In cases where pulmonary consolidation is primary, the signs are those of interstitial pneumonia, emphysema, tuberculosis and obstruction from compression. When the dilatation is deep seated, frequently the physical signs appear normal or perhaps only show some harshness of the respiratory murmur, thus illustrating very aptly how completely the physical signs of bronchiectasis may be masked.

Diagnosis.—The peculiar nature of the sputum and its persistent paroxysmal expulsion are the chief evidences which may be regarded as distinctive. In a large proportion of cases it is not possible to recognize bronchiectasis, either from want of definiteness in its classical manifestations or because the symptoms of the associated primary affection predominate.

A sacculated bronchiectatic cavity may be distinguished from one due to pulmonary tuberculosis by the continued absence of tubercle bacilli and of elastic fibre in the sputum, the absence of fever and the temperature curve peculiar to tuberculosis. Moreover, in bronchiectasis the cavity is usually located near the centre of the lungs, and the condition, though persistent, is not attended with signs of progress. In tuberculosis, on the other hand, the cavity is near the apex and the condition is markedly progressive. The observation of Stokes, that in tuberculosis there is first dulness and then cavity formation, and in bronchiectasis, first cavity then dulness, is important to bear in mind.

Small basic or circumscribed empyema emptying into the bronchi may be mistaken for bronchiectasis. There is, however, frequently in these cases the history of acute illness with a sudden exacerbation, or there is a gradual decline of health with dyspnœa, especially on exertion. In both conditions there is profuse expectoration of muco-purulent matter in large quantities. In both also, if the empyema is of long standing, there may be clubbing of the finger nails. The diagnosis in these cases is very difficult. The history of the case in many instances must be most carefully and minutely inquired into in order to enable a differentiation to be made.

Abscess of the lung may be differentiated from bronchiectasis by the history of a succeeding illness, such as severe pulmonary hæmorrhage and traumatism. Gangrene of the lung, on account of the great fetor of the breath attending it, may be sometimes confused with bronchiectasis. Differentiation may, as a rule, be easily established by the absence of acute conditions which usually precede gangrene, such as pneumonia or embolism, and of the characteristic sputum and cough. Moreover, with gangrene of the lung the constitutional symptoms of prostration are much more profound and appear acutely.

Actinomycosis may also simulate bronchiectasis. Its diagnosis may be made by examination of the sputum and the recognition of the actinomycetes.

Prognosis.—The condition may exist for a long time, and while there is some embarrassment of the circulation, it permits of a certain amount of physical activity. Its general tend, however, is toward a fatal issue. Treatment may not infrequently succeed in checking the amount of secretion and its decompositton and the patient enjoy a moderate degree of good health for several years. When it occurs in children as the result of measles or whooping-cough, recovery may occasionally be observed.

Treatment.—It is apparent that remedies can have no effect upon the structural changes incident to bronchiectasis and, in many instances, little upon those affections which lead to its development. The line of medication which is productive of the best results is that of antisepsis. Cough mixtures and sedatives are to be condemned. The diminution and removal of the secretion, together with its deodorization when fetid, should be the aim. For this purpose those remedies recommended for fetid bronchitis and pulmonary tuberculosis should be employed. When there are no indications of tendencies to hæmorrhage, creasote vapor may be used. The patient being placed in a small room, with the eyes protected and nostrils plugged, is allowed to inhale the fumes of commercial creasote as it arises from a vessel placed on a spirit lamp. At first the vapor is irritating, but a tolerance is soon established. Commencing with inhalations of fifteen minutes every other day, the time should be gradually increased until the inhalations are taken hourly every day. This treatment should continue for three months.

Intra-laryngeal injections are efficacious. A drachm of the following may be injected with a suitable syringe twice a day: Menthol, 10 parts; guaiacol, 2 parts ;, olive oil, 88 parts. A diminution of fœtor and general improvement will follow. For other intra-laryngeal injections see Laryngeal Tuberculosis.

General remedial treatment includes tonics for the general health and the use of guaiacol, creasote, salol, eucalyptus, terebene, naphthol and remedies of this class, and hepar sulphuris, calcarea in trituration or in the form of the hypophosphites, sulphur, stannum, and silicia, according as their special indications exist.

Coughing "up-hill," as it is called, will facilitate the expulsion of the secretion. That is, during the paroxysms of coughing the patient is to lie with the head lower than the trunk, in order to avail himself of the assistance of gravity.

Surgical measures in the form of drainage and direct injection of antiseptic fluids into the cavity have been attended with good results in some instances. Their use should be confined to cases where the cavity lies near the surface and where the patient is in good condition.

ASTHMA.

Asthma is an affection characterized by paroxysmal attacks of dyspnœa attended with a sense of præcordial constriction and suffocation due, probably, to spasmodic contraction of the muscular fibres of the bronchial tubes and hyperæmia and turgescence of their mucosa. It is generally agreed that the affection is a neurosis. The term, asthma, is employed both in a generic and qualified sense, hence the expressions bronchial, cardiac and renal asthma, but modern nomenclature inclines to limit its application to that condition which depends upon disturbance in the bronchi.

While it is generally agreed that an asthmatic paroxysm is at-a narrowing of the bronchial tubes, there is some pinion as to how this phenomenon occurs. The principles which have been offered in explanation are: (1) that t spasm of the muscular fibres of the bronchi, due to irritation; (2) that it is the result of hyperæmia of the ucosa of vaso-motor origin (Traube, Weber); (3) that l form of inflammation of the smaller bronchial tubes us described by Curschmann and called by him *bronchiolitis*; (4) that it is a tonic, spasmodic contraction of the diantrich and Bamberger) arising from super-excitation of e phrenic nerve resulting in a [illegible]tial inhibition of the action of the diaphragm. Other theories a[illegible] those of spasm of the inspiratory muscles (Budd, Kidd, Germa[illegible] Sée, Steavenson), paralysis of he bronchial muscles leading to loss of expiratory power (Walshe), fluctuating hyperæmia analogous to urticaria (Sir Andrew Clarke), and a special microbe (Bee, Bean, Traube).

While these several theories have more or less substantial claims for consideration, the first, namely, spasm of the bronchi from nerve irritation, has by far the greater number of advocates and may be said to be that generally accepted. It is also probable that hyperæmia, turgescence and inflammation are factors in some cases, but scarcely so in those where there is no catarrh or where the asthmatic paroxysm disappears suddenly.

Another question is that of the identity or relation of asthma to vaso-motor rhinitis or hay fever. Sir Andrew Clark suggested that if the paroxysmal phenomenon, which takes place in vaso-motor rhinitis, occurred in the bronchial tubes, making due allowance for anatomical differences, asthma would arise. It is claimed by some observers that the two affections are essentially one and the same, hay fever being a vaso-motor paresis of the walls of the blood-vessels lining the nasal cavity and asthma a vaso-motor paresis of the vessels lining the bronchi (Bosworth). Inasmuch as the pathological changes in hay fever are non-inflammatory, this statement will at once be seen to be at variance with the observations of Curschmann. This difference may be explained by the fact that inflammatory action occurs in some forms of asthma, while in others it is absent—in short, the affection presents distinctly different types.

Ætiology.—The origin of asthma may in general be said to be attributed to constitutional tendencies and reflexes, but in not a few instances it remains obscure. As in other neuroses, many diverse influences and conditions are encountered. Predisposition is observed in heredity, the disease seeming to prevail more in certain families, especially those inclined to nervous affections. It is also more frequent in men than in women, in the proportion of two to one, and in those well placed in life than among the poor. Age does not appear to be a factor, as it occurs in both infancy and old age; the greater number of patients, however, are under forty. As far as season is concerned, asthma is more frequently observed in damp, cold and changeable weather. Among dyscrasias gout occupies a prominent position and its relation to asthma in some cases is clearly recognizable. The affection may also depend upon anæmia, rheumatism, syphilis and scrofula. Again, it not infrequently follows an arrested pulmonary tuberculosis from the pressure of an enlarged gland on the vagi.

In some patients "taking cold" is commonly followed by an attack of asthma, the incident bronchitis being the excitant. In others change of climate brings on an attack. In some instances gouty manifestations may alternate with symptoms of asthma. Affections of the naso-pharynx are also a very frequent cause.

The exciting causes may be conveniently divided into the direct and reflex.

Direct Causes.—These include acute bronchitis, inhalation of irritants, such as smoke, fog, dust, the pollen of certain plants, the emanations of certain animals, and whooping-cough. In some cases the bronchitis may be of the form described by Curschmann as mentioned.

Reflex Causes.—Here are encountered chronic inflammations and irritation in some other local affection, more especially the nasal passages, where hypertrophic rhinitis and polypi play an important part; irritation of the ovario-uterine and genital tract, mental emotion, such as worry, fright or depression, pressure upon the pneumo-gastric nerve, swollen glands, particularly the bronchial; gastric irritation from over-eating and the ingestion of certain articles of food; suppression of eruptions, intestinal irritation and irritation of the cord near the medulla.

In all instances it must be taken into consideration that these factors are generally engrafted upon peculiarities of constitution.

Asthma arises secondarily in connection with Bright's disease (uræmia), disease of the heart, especially mitral insufficiency, and emphysema. Long continued asthma is liable to develop emphysema which, in turn, may act as an excitant of the asthmatic paroxysms.

Morbid Anatomy.—There is, strictly speaking, no known morbid anatomy in asthma, for whatever may be the condition of the bronchial tubes and their mucosa during an attack there are no records of post-mortems of persons who have died during an asthmatic paroxysm. In the instance of death of an asthmatic, at other times the pathological changes indicative of other affections, such as emphysema, chronic bronchitis and cardiac dilatation, are alone found. It is chiefly for this reason that the ætiology as well as the morbid changes which occur in this affection have been the subject of so much discussion.

Symptoms.—Asthma may be regarded as appearing in three general forms: the spasmodic or bronchial, the bronchitic, and the so-called hay asthma. Bronchial asthma is that variety which is often described as pure spasmodic asthma. The phenomena appear without any very marked symptoms of bronchitis or sensitiveness to pollen or emanations. The affection is distinguished by the sudden appearance of the paroxysm, the peculiar dyspnœa, and the absence of all traces of morbid conditions in the intervals.

The paroxysms most always come on at night after the patient has been asleep for a while, having perhaps retired in apparent good health. In some instances there may be prodroma, such as a sense of præcordial constriction, nervous or gastric irritability, mental depression, passage of large quantities of urine and sometimes languor and drowsiness. The patient suddenly awakes with the sense of great oppression. Soon the dyspnœa may become intense. The respiratory efforts are slow and labored, sometimes attended with audible wheezing. The inspirations are long drawn out, indicating that the narrowed bronchial tubes prevent the air from entering. The expirations are even more difficult. The auxiliary respiratory muscles are all called into operation; the abdomen is hard and contracted. The position is that of orthopnœa and the patient makes violent efforts to breathe. Sometimes he may sit on the side of the bed, seize the back of a chair for support and by fixing the shoulders call the extraordinary muscles of respiration into action. The number of respirations are not much accelerated. The countenance is anxious, pale or cyanotic. Speech is monosyllabic and the surface of the body bathed in cold perspiration. The pulse is quickened and thready and the temperature normal or below the normal point.

Cough is a variable symptom; in some cases it is absent, in others present, but not to a marked degree. In some cases it is more permanent, at first light and dry, with difficult expectoration.

After the obstruction has lasted for a while and the patient seems to be at the limit of endurance, the breathing becomes easier and frequently with a paroxysm of coughing relief comes, and the patient exhausted falls asleep. This respite may, however, be only temporary and a second attack comes on.

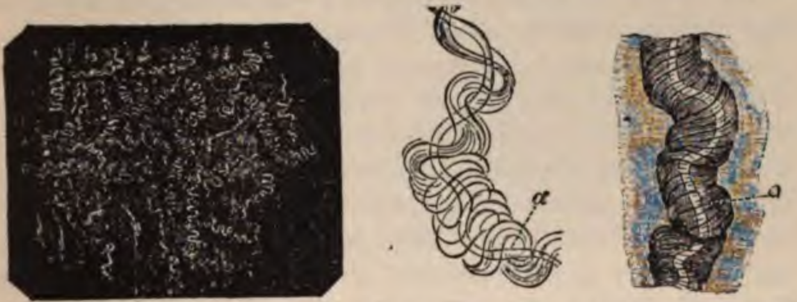

FIG. 39.—Curschmann's spirals. (After Curschmann; Tyson.) The first or dark figure represents the natural size. Figures, *a*, represent the central thread enlarged.

The sputum in asthma possesses marked peculiarities. It is non-aërated, extremely viscid and scanty and is raised with difficulty, coming up in yellowish or grayish-yellow, sago-like masses, called the "perls" of Laennec. Microscopic examination shows these bodies to be made up of masses of spiral fibres, called "Curschmann's spirals," being first described by Curschmann. The spiral portion consists of a mass of fibres so interwoven as to form a tube-like body. They are found on further examination to contain cells which have undergone fatty degeneration, pus cells and cells from the bronchial mucous membrane. These bodies, however, are not peculiar to asthma alone, but are present in fibrinous bronchitis and pneumonia. They are caused, according to Schmidt, by the movements of the air in the bronchial tubes during violent paroxysms of coughing or dyspnœa. In addition there are acicular crystals, called the Charcot-Leyden crystals, which were first discovered by Leyden and subsequently also found by Charcot in leukæmia, the spleen, marrow and semen. A second form of Curschmann's spiral is also found. These consist of tightly curled coils of mucin fibrils, in the centre of which there is a bright thread of what is probably transformed mucin. These are supposed by Curschmann to be the product of bronchiolitis. The sputum may also contain crystals of calcium oxalate and phosphate. The blood shows great increase of eosinophiles.

The course of asthma is extremely variable. The paroxysm may continue from two to six hours. In some the relief is only partial, and labored breathing lasts for several days. In the more severe forms the attacks may continue for three or four nights, while during the intervals there may be more or less bronchial irritation, wheezing respiration and cough. The attacks may differ very much in severity. In recent cases they may be more of a nervous type, while in those who have had repeated attacks the paroxysms are more severe, and in time emphysema or chronic bronchitis develops.

The bronchitic form, as the term implies, is that more or less intimately associated with bronchial inflammation. The relation of bronchitis to asthma is that of both cause and sequela. There are some varieties of asthma where an attack of bronchitis is distinctly the exciting cause. Again, in many instances as a result of hyperæmia, bronchitis follows an asthmatic attack. Finally, in chronic bronchitis patients may suffer from attacks of spasmodic contraction of the bronchi. It is in this form that emphysema is especially prone to develop, and which when once established may react in turn as an exciting cause in itself.

Hay asthma, or, as it might better be called, pollen asthma, is that variety which accompanies hay fever or vaso-motor rhinitis. In addition to the symptoms which characterize that condition the bronchial mucous membrane may manifest a remarkable degree of sensitiveness, and attacks of asthma with cough and præcordial constriction, loud wheezing, and in some cases a more or less abundant mucous expectoration, suddenly develop. The manifestation of these symptoms depends entirely upon the presence of the exciting cause in the atmosphere. The excitant may be the pollen of hay, roses, golden rod or some other plant. Many persons know by experience when to expect the attacks and seclude themselves or leave for some immune locality. The emanations of certain animals is said to have a similar effect, but these conditions are doubtless sometimes hysterical.

In some forms of hay asthma the rhinitic symptoms predominate and in others the asthmatic; in others again the two are blended.

While asthma in itself cannot be regarded as a serious affection, its consequences when long continued may be eminently so. Emphysema and chronic bronchitis develop, and in time the right side of the heart dilates and all the phenomena of cardiac dilatation with venous stasis, etc., may occur.

Physical Signs.—*Inspection* shows the chest to be in a condition of over-distention or fixed expansion. This is due to the fact that with each inspiration the amount of residual air is increased, while expiration is obstructed. Inspiration is short and quick, while expiration is greatly prolonged, being four or five times as long as inspiration.

The ordinary respiratory movements are absent; the thorax moves up and down, but there is no lateral motion. The auxiliary muscles of respiration are called into play and the abdominal muscles, especially the recti, are contracted, tense and hard, the pressure sometimes being sufficient to expel the contents of the bladder and lower bowel. Notwithstanding the contraction of the diaphragm with each respiration there is sinking in of the epigastrium and in severe cases in spite of the distention of the lungs, of the supra- and infra-clavicular spaces. In spite also of the great dyspnœa the respirations are not increased, but, on the contrary, they may sometimes be less frequent than normal.

Palpation shows rhonchial fremitus, while vocal fremitus may be diminished by the bronchitis and further obscured by emphysema. Palpation, however, is of negative practical value. *Percussion* may not yield any sign of change, but frequently there is higher resonance and in old cases semi-tympanitic resonance. The superficial area of cardiac dulness may be diminished or apparently obliterated. The liver is depressed.

Auscultation shows that the ordinary vesicular murmur is absent or is audible only over limited areas, being displaced by an ever-changing variety of dry râles, sibilant and sonorous. These râles include a variety of sounds, wheezing, whistling, cooing, etc. Toward the end of the attack they become moist. The whole of both lungs may display these signs, but sometimes only one. The loss of the vesicular sound is probably due in part to its obscuration by the loud rhonchi, to the obstruction of the bronchioles from the narrowing of their walls and the presence of the viscid mucous secretion.

In cases where the paroxysm terminates suddenly the influx of air into the lungs may give rise to puerile respiration. After the subsidence of the attack in recent cases the transitory compression disappears and the lungs return to their normal condition. In old cases, on the other hand, emphysema develops.

Diagnosis.—The diagnosis of asthma is generally free from any difficulty. There are, however, certain affections which may sometimes simulate it to a greater or less degree. The most notable of these are cardiac dyspnœa, emphysema, spasm of the glottis, tracheal stenosis, hysterical dyspnœa, spasm of the diaphragm, uræmia and embolism of the pulmonary artery or its larger branches. The dyspnœa dependent upon disease of the heart, while it resembles that of asthma, differs in the following particulars : It comes on after exertion and mental excitement, is sighing, and rhythmatically accelerated ; the expiratory act is not prolonged ; there are also the physical signs of cardiac disease. There are cases, however, where true spasmodic asthma may be engrafted upon cardiac disease. This condition is especially prone to appear in the morning and is one of very great suffering.

Emphysema is distinguished by the fact that the dyspnœa is of a different type, being more or less constant and much increased by exertion. Moreover, there are changes in the lungs and heart which characterize the disorder as indicated by the physical signs.

The dyspnœa of spasm of the glottis is distinguished by the prolonged and forcible character of the inspiration, the comparatively easy expiration, the laryngeal stridor and characteristic croupy cough. The same features characterize croup, œdema of the glottis and tracheal stenosis. It is important to bear in mind in this connection the observation of Biermer, that in all affections causing narrowing of the larger air-passages the dyspnœa is chiefly on inspiration, while in that dependent upon narrowing of the small bronchi it is chiefly on expiration.

In croup, furthermore, the head is thrown back and extended, and in the severe forms the respiratory efforts of the patient, though violent, only partly fill the lungs and in consequence there is falling in of the supra-clavicular spaces, lower part of the sternum and adjacent costal cartilages and retraction of the abdomen. During expiration, which is quick, these parts resume their natural condition. In asthma, on the other hand, the head is in a forward position, the thorax is expanded, the epigastrium sunken in during inspiration and the abdomen is hard and retracted during expiration. Moreover, the characteristic physical signs are present.

Paralysis of the posterior crico-arytenoid muscles, like croup, is attended with inspiratory dyspnœa. The stridor and the close approximation of the laryngeal cords in inspiration, as shown by microscopic examination, are conclusive evidences of this condition. Another characteristic symptom is that the dyspnœa, unlike asthma, does not come on in paroxysms.

Hysterical dyspnœa is to be distinguished by the presence of rapid breathing, which may continue for hours at a time at the rate of thirty or over per minute, but without any evidence of sibilant or sonorous râles and without any other symptom except, perhaps, those significant of hysteria.

Spasm of the diaphragm, which is a rare affection and one usually associated with hysteria, is characterized by a short inspiratory movement which calls all the muscles of respiration into action, by sinking in of the more yielding portions of the thorax which remain fixed for a few seconds, when there follows a sudden and powerful effort at expiration, accompanied by a sound like hiccough, then another inspiration with the same phenomenon.

Uræmic dyspnœa may sometimes assume the form of a true spasmodic asthma, but the history of the case and the examination of the urine cannot fail to remove any source of confusion which may arise.

Embolism of the pulmonary artery or its larger branches is also sudden in its onset and is attended with great dyspnœa, but is scarcely likely to be mistaken for asthma from the fact that the pulse is thready and irregular ; there is more or less pulmonary œdema, the cardiac impulse is weak and the expression one of excessive anxiety.

Prognosis.—The outlook in asthma must be viewed from the standpoints of recovery from the attacks and the liability to their recurrence. As far as the former is concerned, notwithstanding the great distress and alarm which may be present, the prognosis is favorable, as death seldom if ever occurs. Recovery from the tendency to repetition of asthmatic paroxysms, however, is another matter and depends upon age and the possibility of removal of the cause. In children recovery may be complete, but when the affection occurs late in life the patient may suffer from occasional attacks for the remainder of his days. At the same time there is always a possibility that recovery may take place if there have been no organic changes in the lungs or heart.

An important feature in long-standing cases is the permanent structural changes in the lungs and heart which often follow and which may lead to the most disastrous results. At the same time the patient may be subject to attacks of asthma from early childhood and live to old age.

Treatment.—The treatment of asthma consists of the relief of the paroxysms, the care of the patient during the interval of attacks and the removal of the cause. The great number of nostrums on the market for its cure are evidences of how unsatisfactory treatment frequently proves and at the same time how much it is abused.

TREATMENT OF THE PAROXYSMS.—Whenever it is possible the use of sedatives should be avoided. Frequently, in recent cases a well selected remedy will accomplish the desired result, but in many others this is not possible, especially in old cases and where remedies of the sedative class have long been employed. The principal palliatives are as follows :

Nitre paper fumes, that is, bibulous paper soaked in a solution of nitre, ignited after drying and the fumes inhaled. This gives relief in a wide range of cases.

Stramonium leaves made into cigarettes and smoked.

Amyl nitrite affords speedy relief in some instances, but often fails in others.

Nitro-glycerine and *sodium nitrite* are likewise useful. The latter is not so rapid in its action, but is more lasting in its effects.

Lobelia, an old remedy which has fallen into disuse, deservedly so when given in large doses, enters into many of the well-known patent medicines. In minute doses or dilutions it will often act when the attack is of gastric origin.

Chloroform, direct inhalations or its fumes poured on hot water. Ether is often better than chloroform and is certainly safer.

Ethyl nitrite, inhalations, eight to ten drops on a sponge.

Dilute hydrocyanic acid was recommended by MacKenzie and others. Three drops to half a pint of hot water. Inhalation of the vapor will often arrest the paroxysms, especially in cases where neurosis is marked.

Methyl chloride, sprays applied directly on the back; if not successful, light spraying over the upper part of the chest.

Grendelia robusta is an important and useful remedy. Many cases will yield to its influence. It may be administered by the mouth or by inhalation of the fumes. In the latter instance the plant is saturated in a solution of nitre, then dried and ignited. The preparation may also be made into cigarettes and smoked.

Euphorbia pilulifera, an Australian plant, often affords marked relief. It has been employed in half- to one-drachm doses of the tincture.

Morphia, alone or with atropia, in doses of gr. $\frac{1}{4}$ and gr. $\frac{1}{120}$ respectively, subcutaneously, often relieves after all other remedies fail. The danger of forming the morphine habit should be considered.

Cocaine is regarded by some as a palliative of the first importance. It may be applied in solution on pledgets of cotton to the nostrils or with an atomizer and repeated every five minutes until relief is obtained. The danger of forming the drug habit and also of toxic effects of the drug itself must be considered.

Other remedies that should be borne in mind are an emetic, which will stop an attack in some cases; alcoholic stimulants; strong coffee; chloral; bromoform; hyoscine; quebracho; and inhalations of camphor and menthol.

It cannot be said that any of these remedies has any special indication. They must all be used empirically, for every practitioner is mindful of the fact that a drug that has been successful in one case of asthma fails in another apparently like it in all respects. The experience of the patients counts for much and should always receive consideration.

The remedies with special indications which are of service in the paroxysm are the following:

Aconitum, for recent cases where exposure appears to be the exciting cause, especially in young and well nourished subjects, with fever, restlessness and full, bounding pulse.

Bryonia, in recent cases with bronchitis, pain in the side and soreness.

Arsenicum album may be of service for violent paroxysms and during the intervals between the attacks. In the first instance its chief indications are the severity of the symptoms with lividity, frequent, small pulse, loud wheezing and great prostration. In chronic cases with emphysema it will also prove useful.

Cupri arsenis is of value in the bronchial forms of the affection.

Cuprum metallicum, in asthma of a purely nervous origin.

Nux vomica, in those forms dependent upon disorders of the digestive system which is a frequent cause of irritation.

Veratrum album, when the paroxysms are violent, with coldness of the surface, cold perspiration and heat prostration.

Ipecacuanha, in drop doses of the tincture, will relieve some cases with accumulation of mucus in the bronchi, paleness, nausea and cough.

Potassi iodidum, especially in rheumatic subjects, will often produce marked results and is a remedy of importance.

Antimonium tartaratum, in cases with much loose rattling mucus, wheezing and cough. It is useful in both adults and in children.

Other remedies which will prove of service according to their individual indications are ambra, aralia, arsenici iodidum, belladonna, cactus, cannabis sativa, hepar sulphuris, moschus, pulsatilla, sambucus especially in children, tabacum, sulphur, graphites and zincum.

After the subsidence of the paroxysm, bryonia, antimonium tartaratum, ipecacuanha and potassi iodidum are the remedies which will usually be found most effective for the bronchitis which may remain.

Treatment During the Intervals.—A most diligent search should be made to ascertain the cause and if possible remove it. In reviewing the ætiology it will be seen that a wide range of possible influences must be taken into consideration, chief among which is the neurotic temperament. For this condition hydrotherapy, galvanism, massage and change of air should be considered. Examination of the nasal passages should be made in all cases. Swelling of the superior and sometimes the inferior turbinated bones may be found. This should be removed. For this purpose chromic acid in a saturated solution, applied with probes with hollow balls at their extremities, may be employed. The superfluous acid must be removed and the nose then plugged for several hours. During the intervals of the treatment antiseptic sprays should be used.

Careful attention should in all cases be directed to the diet, especially as asthmatics are so frequently inclined to suffer from indigestion. The meals should be regular, and over-eating, late suppers or late dinners avoided, as gastric irritation from an over-filled stomach often excites an attack.

Climate is a very important question, as many are greatly benefited by change of air. It is impossible, however, to speak at all with any degree of definiteness; each case must be a rule unto itself. Strange to say, some patients do better in a smoky, foggy climate, such as that of London; others in a dry, elevated locality; and others, again, by the seashore.

General hygiene, it is needless to say, should be of the best, as to exercise, clothing and habits of living.

BRONCHIAL STENOSIS.

Bronchial stenosis is obstruction of a bronchus from constriction or compression.

Ætiology and Morbid Anatomy.—Stenosis due to constriction arises from contractions of healed syphilitic ulcerations, malignant growths which involve the calibre of the tubes, neoplasms within the bronchi, such as polypi and foreign bodies accidentally introduced.

The calibre of small bronchi may become diminished by thickening of their walls in tuberculosis, glanders, syphilis and certain acute inflammations, but scarcely fall under the consideration of the subject in question.

Stenosis from pressure occurs in enlargements of structures within the thorax and neoplasm and is observed in association with aneurysm, solid tumors, abscess of the mediastinum or lungs and extensive pleural effusion.

The effect of bronchial stenosis is not only the arrest or partial obstruction of the entrance and egress of air to and from that portion of the lung beyond the point of constriction or compression, but also the retention of secretions which may be augmented by the irritant effect of the obstructive influence. Sometimes there may be a yielding at the point of occlusion and a discharge takes place, followed by re-accumulation. In many instances the stoppage is complete and collapse of the lung beyond, with subsequent consolidation and possible pleuritic complications, follows.

Symptoms.—The symptoms depend upon the size of the bronchus involved and the extent of the obstruction. Dyspnœa is the most prominent, it is often paroxysmal and when excessive the accessory muscles of respiration are called into action. On account of the imperfect filling of the lung the lower part of the sternum and lower ribs are retracted during inspiration, while expiration is attended with difficulty. Occlusion of the primary bronchus on one side would be attended on inspiration by retraction of the lower part of the chest wall and intercostal spaces upon that side.

When a large bronchus is suddenly closed by some obstruction, dyspnœa is most urgent and death quickly follows. When smaller tubes are involved or when a large tube becomes gradually implicated the symptoms are not so urgent. The dyspnœa may be slight, accompanied by stridor. Spasmodic cough and a thick expectoration are present in such cases. Fever may also develop.

In the efforts of breathing the movements of the larynx are slight. If the lung collapses consolidation follows accompanied sometimes by pleurisy.

PHYSICAL SIGNS.—*Inspection* shows the impaired movement on the affected side with retraction in well marked cases. *Palpation* shows diminished or absent fremitus. *Percussion* remains unaffected for a time, although it is less influenced by inspiration and expiration than in health. As pulmonary collapse develops dulness is elicited.

Auscultation gives diminution of the normal vesicular murmur on inspiration due to the lessened amount of air entering the lungs and the presence of sibilant and sonorous râles at the seat of obstruction.

Diagnosis.—The recognition of bronchial stenosis is often very difficult. The primary existing condition and the history of the case will furnish important evidence. The presence or absence of sibilant and sonorous râles and retraction of the lower part of the sternum and intercostal spaces during inspiration with difficult expiration are important guides. Laryngeal stenosis may be eliminated by laryngeal examination. In laryngeal stenosis it is important to note that there is marked involvement of the larynx, while in bronchial stenosis this is not the case.

Prognosis.—The duration of the condition is indefinite. It is generally progressive and the outlook correspondingly bad, especially if a large bronchus be the seat of obstruction. When due to the accidental lodgment of a foreign body the prognosis obviously depends upon its removal. When a large bronchus is suddenly occluded death from apnœa speedily ensues.

Treatment.—In all cases the cause must be ascertained. When taking the ætiology into consideration it is apparent that in a large number of instances, such as solid tumors of the mediastinum, aneurysm of the aorta, etc., treatment must be palliative. When associated with syphilis antisyphilitic treatment should be instillated. Foreign bodies have sometimes been removed by emetics. For further consideration of the subject the reader is referred to works on surgery.

BRONCHIAL CONCRETIONS.

Concretions are in rare instances found in the bronchi. They are formed by the inspissation of secretions, especially in bronchiectatic cavities. They are usually small, but some may be as large as hazelnuts. They are composed principally of carbonate and phosphate of lime. Very rarely masses of bone or cartilage separated from outgrowths of the bronchial cartilages are found.

In some instances there are no symptoms at all, in others there may be those of pulmonary disease with hectic.

There are no distinctive signs. Diagnosis is impossible.

SECTION II.

DISEASES OF THE LUNGS.

CONGESTION OF THE LUNGS.

Pulmonary congestion occurs in two forms, the active and the passive.

(I) Active Congestion of the Lungs.

This term implies a condition of hyperæmia of the pulmonary structures arising independently of inflammation, that is, the pathological changes stop short of such a condition, and are not necessarily associated with it. It must in this aspect be regarded as a primary process. This view, however, has not been accepted by all, especially the Americans and English. The French, on the other hand, call it "*maladie de Woillez*," in the sense of a primary affection.

Ætiology.—The causes which may excite an abnormal afflux of blood to the lungs are exposure to excessive heat or excessive cold, the ingestion of large amounts of alcohol, inhalation of irritant gases, violent efforts at inspiration with closure of the glottis and consequent diminution of atmospheric pressure within the chest, as in laryngismus stridulus, pertussis and croup, and violent muscular exertion, and intense mental excitement when the heart is not strong and is excited to wildest action. The condition also follows embolism and inflammation of structures adjacent to the lungs.

Pulmonary congestion is likewise associated with acute inflammations of the chest, such as bronchitis, pneumonia, pleurisy and acute pulmonic tuberculosis. It is also encountered in chronic tuberculosis. In such instances the condition is secondary to the prëexisting disease, and is of quite frequent occurrence, while the type of pulmonary congestion in question, namely, that arising independently of some other morbid state is unusual.

Collateral hyperæmia occurs when the opposite lung or some other part of the body becomes anæmic through obstruction of the circulation of some other part.

Morbid Anatomy.—The affection is generally bilateral and confined to the base of the lungs which are practically in the same condition as in the first stage of inflammation. The involved areas are dark red in color, the vessels are engorged with blood, and the bronchial mucosa injected. On section, a frothy, bloody fluid exudes indicating œdema, which is present to a greater or less degree in all forms of acute congestion. The air-cells, the vessels of which are distended, may show evidences of catarrh and the presence of blood.

In cases of embolism there may be rupture of the blood-vessels and hæmorrhage into the tissues. Minute points of extravasation may be found without hæmorrhage.

The lung retains its normal crepitancy and power to float when thrown into water, but it contains less air than normal on account of the engorgement of the vessels which necessarily encroaches upon the calibre of the air-cells and finer bronchi.

Symptoms.—There may be an initial chill. Fever is usually moderate, that is, from 101° to 103°, but in some cases may be higher. There is very decided dyspnœa. If the congestion involves much of the lung it is excessive. There are sticking costal pains, cough with serous, frothy, bloody or rusty colored sputum, or free hæmorrhage. The initial chill, dyspnœa and cough with bloody sputum are the most prominent of the clinical manifestations, which are by no means clearly defined, and which are strongly suggestive of pneumonia.

PHYSICAL SIGNS.—*Inspection* shows increased frequency of respiration. *Palpation* may reveal slight increase of fremitus. *Percussion* is usually negative, or elicits slight dulness. *Auscultation* shows enfeebled respiration, sometimes a bronchial respiratory murmur and subcrepitant râles.

Diagnosis.—The presence of a given cause, dyspnœa, cough, frothy, bloody sputum, in the absence of much fever, point to pulmonary congestion.

It is apparent that at the outset of the above symptoms it is impossible to tell whether the condition is that of the first stage of a not very pronounced form of pneumonia or simply pulmonary congestion. The only positive means of differentiation is to await the development of subsequent symptoms. Resemblance to pulmonary oedema is also marked. The history of the case, however, usually prevents error.

Prognosis.—The condition usually lasts but for a short time and terminates favorably in a few hours or a few days. In cases where the engorgement of the lungs has been very general and intense, death has occurred. The liability of the condition to run into pneumonia must also be considered. Therefore, notwithstanding its general favorable outlook, acute pulmonary congestion must always be regarded as serious.

Treatment.—The cause should be ascertained, if possible, in all instances, and the treatment modified accordingly. Place the patient in bed, apply dry cups freely, or sinapisms over the chest, also heat to the extremities or a hot foot bath. The bowels should be kept open and the diet light. All stimulants must be forbidden. Venesection has been recommended, especially in primary cases and plethoric subjects, but the following in conjunction with the above general recommendations will render the abstraction of blood unnecessary.

Aconitum, indicated when there is restlessness, anxiety, fever, hacking cough, expectoration of bloody sputum, high bounding pulse and pressing, burning pain in chest, especially in persons of full habit.

Veratrum viride, when there is intense congestion without restlessness, which usually calls for aconite.

Belladonna, marked pulmonary hyperæmia with the face very much flushed, throbbing carotids, pupils dilated, symptoms appearing suddenly.

Ferrum phosphoricum proves of service in those cases when the onset is not so abrupt as in those calling for aconite, veratrum viride or belladonna. There is a tendency for the fever to run high. There is also very bloody sputum. The type of cases where ferrum phos. should be employed are those in which low vitality and anæmia are more or less pronounced.

Phosphorus is called for when the dyspnœa is extreme and pulmonary œdema threatened, in persons of the tall, slender, blonde type inclined to cachexia.

Antimonium tartaratum, when pulmonary œdema is threatened or when there is dyspnœa with suffocative paroxysms.

II. Passive Congestion of the Lungs.

This form of congestion presents two varieties, the hypostatic and the mechanical or obstructive.

1. HYPOSTATIC CONGESTION. HYPOSTATIC PNEUMONIA. SPLENIZATION.

Hypostatic congestion of the lungs is a condition which arises when the circulation through the lungs is so feeble that the blood settles in the lower portion through gravity. Hypostatic pneumonia and splenization are advanced forms of hypostatic congestion, and although regarded by many writers as synonymous with it, strictly speaking, are not so.

Ætiology.—Any protracted illness which necessarily depletes the system, and confines the patient to bed for a long period, furnishes the causal factors for hypostasis of the pulmonary circulation, which are, feebleness of heart action, depraved states of the blood and gravity. The first two are the most important, for a healthy person may remain in bed for a long period without developing hypostatic congestion. Thus, the condition may be looked for in protracted fevers, such as typhoid, acute articular rheumatism, cancer, tuberculosis and illness of any kind in the aged. It may also follow surgical operations and is quite frequent in certain diseases of the nervous system, such as hæmorrhages and tumors of the brain. It has occurred during the first two or three weeks in cases of hæmorrhage into the medullary pia mater, fracture of the cervical vertebræ and ascending paralysis, its presence being demonstrated by post-mortem examination. It may likewise follow carbonic dioxide gas inhalation and poisoning by opium and other narcotics.

In cases where the patient is in a semi-comatose condition hypostatic congestion may arise in association with areas of consolidation, due to aspiration pneumonia from the lodgment of particles of food in the air passages which may occur in ineffectual attempts at swallowing.

Morbid Anatomy.—The lower and posterior portions of both lungs are the seat of the morbid changes which do not follow the conformation of the lobules, but rather the line of gravity as the blood settles, thus the lungs are not equally involved. The tendency of the process is upwards on this line. The lung is dark red, heavy and in more acute cases infiltrated with serous exudate and blood. The tissues are firm to the touch and a cut portion sinks when thrown into water. Microscopical examination shows that the alveolar blood-vessels are greatly distended and project into the alveoli which contain red and white blood-corpuscles. The interstitial tissues are also infiltrated with red and white blood-corpuscles. In the later stages the red corpuscles either rëenter the circulation or break down and form pigment granules with the alveolar epithelial cells.

Hypostatic pneumonia commences as an intense hypostatic congestion and œdema. The process, however, advances to a form of inflammation in the terminal bronchi and air vessels, catarrhal in nature, but more fibrinous and diffuse than the ordinary form of broncho-pneumonia, and accompanied by a hæmorrhagic œdema. The dependent portions of the lungs posteriorly are commonly involved. The process occurs as a terminal complication in many diseases.

In long continued fevers hypostatic congestion frequently passes into hypostatic pneumonia. Some of the changes observed in hypostatic congestion are no doubt post-mortem, but, as pointed out Piorry, it is none the less a distinct lesion.

Adjacent to the areas of hypostatic congestion and pneumonia there is always more or less œdema.

Splenization is another condition which may arise as the result of congestion. It occurs when the congestion is accompanied by an escape of blood into the lung tissue, and a serous effusion into air-cells. The lung tissue itself becomes saturated with blood, and the serous effusion excessive. The result is a soft pulpy mass like the spleen, hence the name.

Symptoms.—In many cases there are no symptoms, and the condition may exist without recognition. On the other hand, when pronounced, evidences of defective aëration of the blood may be noted in cyanosis and perhaps increased rate of respiration. Excessive prostration is frequently a prominent symptom, and in the aged, sleeping with the mouth open is said by some observers to point to hypostatic congestion.

In all cases of protracted illness, and conditions which confine the patient to bed for a long time, frequent examination of the chest should be made.

PHYSICAL SIGNS.—There is some increase of vocal fremitus, slight dulness on percussion, feeble respiratory murmur, sometimes bronchial in character, and moist râles. All these signs are situated at the base of the lungs posteriorly, and are the only evidences of the condition. Bronchial respiration and râles denote a more decided grade of the condition, viz., hypostatic pneumonia.

Prognosis.—Hypostatic congestion must always be regarded as an evidence of depressed vitality and enfeeblement of the heart action secondary to a primary morbid state, hence its presence is of unfavorable significance. The prognosis, however, depends upon the underlying cause.

Treatment.—The line of treatment should be directed toward the primary condition. In those conditions likely to be attended with hypostatic congestion of the lungs it is important to change the patient's position frequently. Another preventive measure is to cause the patient to sit up when he is able and take long deep inspirations several times a day. General treatment consists of strongly nutritious and stimulating diet, sponging the skin, and the administration of enemata to keep the bowels free. Special treatment directed to the general condition should likewise include the condition of the lungs. The following are suggested with this end in view, acidum muriaticum, arsenicum album, rhus tox., antimonium tartaratum and phosphorus.

When the heart action seems especially at fault, digitalis, strophanthus and strychnia should be considered.

2. Mechanical or Obstructive Congestion. Brown Induration of the Lungs.

This condition is a chronic change of the lungs associated with induration, proliferation of connective tissue and pigmentation, the result of obstruction of the blood-flow from the lungs into the heart. It may always be traced to some slow-acting obstructive influence.

The principal cause and that responsible for the greater proportion of all cases is organic disease of the heart, namely, mitral stenosis, mitral regurgitation, aortic regurgitation when the mitral valve has secondarily failed, and degeneration of the heart walls. Of these mitral stenosis is probably the most prominent.

Aneurysm of the aorta and neoplastic growths situated in the thorax may produce the same effect. The condition may likewise arise in association with cerebral diseases and injuries.

It is most frequently observed in those under middle age.

Morbid Anatomy.—The lungs are enlarged and of a dark reddish-brown color. The air-cells crepitate but little. On section it is seen that the lung substance is hardened and resists the efforts to cut or tear it.

The loss of elasticity is to a certain extent compensated for by hypertrophy of the muscular fibres which surround the infundibula. Another feature is that the cut surfaces change rapidly to a bright red. This is due to oxidation of the hæmoglobin when exposed to the atmosphere. Microscopical examination shows proliferation of the connective tissue of the lung, which is essentially a feature of chronic obstructive congestion or brown induration, in contradistinction to hypostatic congestion where it is absent. There is also chronic congestion and distention of the capillaries and thickening of the alveolar septum in which embryonic tissue and additional fully formed fibres can be recognized. There is likewise pigmentation of the inter-alveolar tissues usually in the form of hæmosiderin responding to the usual tests of iron, and catarrh of the alveolar cells in which the epithelia are desquamated, pigmented and in all stages of alteration. Catarrh of the bronchial mucosa and some œdema is also present. In some instances consolidated patches due to hæmorrhages may be observed. These vary according to age.

The process commences at the extreme base of the lung, extends upward and ultimately involves the whole lung uniformly.

Symptoms.—Dyspnœa and cough with expectoration of a frothy serous and bloody sputum containing pigment alveolar cells are the most characteristic symptoms. Free hæmoptysis is not uncommon. In addition there are symptoms common to organic heart disease with weak compensation, except in the rare instances of other possible primary conditions mentioned in the ætiology, when the clinical features will be modified accordingly.

Physical Signs.—These are not very definite. There is dulness on percussion at the base of the lung, and general feebleness of the respiratory sounds which are broncho-vesicular.

Diagnosis.—The recognition of the condition depends upon the history of the case, which with rare exceptions is that of organic disease of the heart, and the expectoration of pigmented alveolar cells.

Treatment.—The nature of the primary condition determines the line of treatment. As cardiac disease is responsible for nearly all cases, reference should be made to that subject.

DIFFUSE PULMONARY APOPLEXY.

This term signifies hæmorrhage into the air-cells and lung tissue with or without laceration of the pulmonary parenchyma. It may be caused by rupture of a thoracic aneurysm adherent to the lung or by direct traumatism. The lung tissues are also sometimes the seat of a diffuse hæmorrhagic infiltration in septico-pyæmic and cerebral diseases. In these conditions the lungs may be quite evenly infiltrated with blood and on section present a gelatinous and blackish appearance, the air-cells being replaced by a sero-sangruinolent accumulation and the lacerated area changed into a soft pulpy mass. In some instances hæmorrhage into the lungs may be more circumscribed, and the involved area presents the appearance of an ordinary infarct.

The symptoms of diffuse pulmonary hæmorrhage are excessive hæmoptysis, intense dyspnœa, cyanosis and collapse. Notwithstanding their intensity and extreme gravity they are not distinctive.

The physical signs disclose an extensive consolidation of sudden formation.

The prognosis in these cases is very unfavorable, death may occur in a few moments after the hæmorrhage.

Rest in the recumbent position, the application of ice to the chest, and the administration of remedies mentioned for hæmoptysis is all that can be done. When collapse makes its appearance stimulants will be necessary. Frequently, all efforts are without avail and the patient immediately succumbs.

PULMONARY ŒDEMA.

Pulmonary œdema is an effusion of fluid into the air-cells and interstitial tissues of the lung. It may be either circumscribed or general.

Ætiology.—Circumscribed œdema is met with in all acute inflammations, congestions, abscess, infarction, neoplastic growths, and sometimes in chronic lesions of the lungs. In these conditions the œdema is confined, more or less, to a limited zone surrounding the seat of lesion, and, clinically generally, does not play an important part.

General œdema of the lung is of grave significance, and it is the condition implied when the term pulmonary œdema is employed. Its consideration forms the basis of this article. It is characteristically observed in the presence of prolonged stasis of the pulmonary circulation, as in mitral disease and heart weakness from any cause. It is likewise common in disease of the kidneys and brain, and acute inflammations and infections. Hence it is observed in a large number of morbid conditions where it usually appears as a terminal complication. Cohnheim has said that persons do not die because they have pulmonary œdema, but have pulmonary œdema because they are dying. However this may be, in a large number of deaths, both from acute and chronic diseases, œdema of the lungs is the first indication of approaching dissolution.

In rare instances pulmonary œdema may suddenly arise from vaso-motor relaxation.

The essential pathological factors which enter into the causation of the lesion in question are variations in blood-pressure, changes in the character of the blood and changes in the walls of the blood-vessels.

Increased tension alone does not cause œdema. According to the experiments of Welch, the chief factor is a disproportionate weakness of the left ventricle, which causes the blood to accumulate in the pulmonary capillaries and right side of the heart. This is apparently especially true in terminal varieties. Pulmonary œdema may likewise arise from weakness of the right ventricle alone.

The change in the blood which favors œdema is increase of fluidity or diluted plasma.

The third factor, namely, changes in the walls of the blood-vessels, is impaired nutrition which arises from disturbance of the circulation and innervation of vessels, and results in increased permeability of their cells. This occurs in toxæmias, and is observed in infection, uræmia, cachexias and sepsis.

Morbid Anatomy.—Œdematous lung tissue is heavy, pits on pressure, but does not sink when thrown into water. It has a watery and sometimes gelatinous infiltrated appearance. The latter condition is frequent at the base of the lungs and may be observed throughout. On section there exudes from the air vesicles a clear fluid, occasionally tinged with blood and usually mixed with exfoliated epithelial cells. Œdematous fluid may be also found in the interstitial tissues and bronchi. There is frequently an associated hyperæmia which may vary in different parts of the lung. On the other hand, there may be extreme œdema without overfilled blood-vessels.

The result of these changes is a more or less airless condition of the alveoli, and consequent interference with respiration.

The pleura in most instances is moist, occasionally there may be hydrothorax.

Symptoms.—General pulmonary œdema may develop slowly or suddenly. As in all other secondary conditions the symptoms of the primary disease are present to a greater or lesser degree. Dyspnœa, cyanosis, cough and expectoration are its symptoms, the chief being dyspnœa. Cough with more or less difficult expectoration of a frothy, blood-tinged sputum is usually present, but in terminal forms, especially those of sudden development, it may be very slight.

Cyanosis necessarily follows as the result of defective aëration, growing more pronounced as the œdema becomes more marked. The countenance is anxious and frequently there is great anguish from the sense of suffocation.

Terminal œdema may take several days to develop, or may destroy life in a few hours. Frequently it makes its appearance suddenly and rapidly proves fatal, especially in croupous pneumonia and Bright's disease.

Fever is not a symptom unless the inflammatory form is recognized. Some speak of such a variety as occurring with inflammation and congestion, but it seems to the writer unnecessary to make such a distinction, as the pathological condition and the fever are due to the primary disease rather than to the œdema. The pulse is feeble and irregular.

A rare form of pulmonary œdema is the *acute angioneurotic* variety. It consists of a sudden dilatation of the vessels and transudation of serum into the surrounding tissue. The cause is doubtless a vaso-motor paralysis from the presence of toxines in the blood, the product of defective digestion. The process is akin pathologically to urticaria and erythema, although the pathology is not clear. It may involve any mucous membrane, including that of the respiratory tract. The patient before seizure is usually in good health except for some acute gastric disturbance. The attack continues for a few hours when it subsides, leaving the patient in a nearly normal condition, except for some weakness. The symptoms are those of the ordinary form. There is always great danger. If the attack is severe it may cause acute dilatation of the cavities of the heart from obstruction of the blood in the lungs and rapidly prove fatal.

PHYSICAL SIGNS.—*Inspection* shows increase of the rate of the respiration, which as the condition advances becomes more labored and shallow. *Percussion* gives some dulness, most marked particularly over the lower lobes. *Auscultation* reveals the presence of moist râles, crepitant, subcrepitant and bronchial, which increase in extent and intensity as the œdema advances, and ultimately involve the whole lung.

Diagnosis.—The gradual or sudden development in the course of any disease, more especially pulmonary, cardiac, infectious and cerebral, of rapid breathing, dyspnœa, cyanosis and a profusion of moist râles of the character described, render the recognition of pulmonary œdema unmistakable.

Prognosis.—General pulmonary œdema is always grave. When it arises from cardiac, infectious, or acute pulmonary diseases, it rapidly destroys life. Circumscribed pulmonary œdema, unless there is a tendency to extension, does not exert an important bearing upon the case, but when it threatens to extend the outlook is serious.

Treatment.—The primary disease necessarily governs to a greater or less extent the line of treatment. In cases of impending œdema of the lungs, the position of the patient should be frequently changed in order to obtain the benefit of gravity, and the bowels should be kept free. It must always be borne in mind that the œdema is always dangerous, and that it may quickly destroy life by failure of the heart from over-distention of the right ventricle. When weakness or disease of the heart is the exciting cause, digitalis, caffein, strychnia should be administered in full doses, preferably hypodermically. In sudden cases when life is threatened six to eight ounces of blood should be withdrawn to relieve the right side of the heart. Dry cups on the chest posteriorly and anteriorly are often of service, also turpentine stupes applied alternately to the back and sides of the chest. Heat should be applied to the extremities. Aromatic spirits of ammonia in thirty drop doses, and ammonium carbonate in three-grain doses are also recommended.

In pulmonary œdema arising in connection with Bright's disease, produce free diaphoresis by giving pilocarpine hypodermically in one-tenth to one-quarter grain doses. Hot packs may also be applied.

In all instances where time permits free catharsis, by the administration of mercurius dulcis, elaterium or magnesium sulphate, should be established.

Other remedies which will prove of service according to the prevailing indications are as follows:

Antimonium tartaratum is useful in acute forms associated with inflammatory conditions of the lungs with intense dyspnœa, loud coarse rattling râles indicating a large quantity of fluid in the bronchi.

Arsenicum album is indicated in Bright's disease or in disarrangement of the renal function, with anxiety, restlessness and thirst for small quantities of water.

Phosphorus may be considered in œdema associated with pneumonia, with violent cough and blood-stained expectoration depending upon the congestion and inflammation.

Adrenalin chloride has been recommended for the acute angioneurotic form. It is to be sprayed down the air passages with an atomizer. A strong solution should be used.

HÆMOPTYSIS.

SYNONYMS.—*Bronchial Hæmorrhage, Broncho-Pulmonary Hæmorrhage, Pulmonary Hæmorrhage.*

Hæmoptysis signifies the expectoration of blood which arises from hæmorrhage from the mucous membrane of the bronchial tubes, usually the smaller, from eroded vessels in pulmonary ulceration and excavation, and more rarely from the larynx, trachea and larger bronchi. When the source is the bronchial tubes the term bronchorrhagia may be employed.

Ætiology.—As a symptom hæmoptysis is of grave significance, and search of its source is always imperative. This, however, in some instances is unsuccessful.

The morbid conditions which may be attended with expectoration of blood are those which lead to pulmonary hyperæmia and rupture of the walls of the arteries of the lungs and aneurysm. They may have their origin in the respiratory tract or outside of its structures.

I. Sources of hæmorrhage situated in the respiratory tract.

(1) Pulmonary tuberculosis. This is by far the most common source of hæmorrhage from the respiratory tract and the lesion which first suggests itself to the mind when hæmoptysis occurs. Observed early in the disease the blood undoubtedly comes from congestion of the smaller bronchi around the foci of infection. In the latter periods it arises from ulceration of the walls of larger vessels, or from rupture of aneurysmal dilations of branches of the pulmonary artery.

(2) Croupous pneumonia. In rare cases hæmorrhage occurs in the early stages. The blood tinged "prune-juice" expectoration, which usually accompanies the disease, while strictly speaking a form of hæmoptysis scarcely comes under that term as it is usually employed.

(3) Pulmonary infarction. In some cases this lesion may be attended with hæmorrhage.

(4) Traumatism of the lungs. Wounds of any kind which lacerates or punctures the pulmonary parenchyma are usually attended with hæmoptysis.

(5) Bronchiectasis. Hæmoptysis is occasionally observed with this condition.

(6) Bronchitis. Acute forms when accompanied by intense types of pulmonary congestion may show a mild degree of hæmoptysis. The fibrinous variety may also induce hæmorrhage.

(7) Inhalation of irritants. Chlorine gas and similar irritants by causing capillary congestion may cause hæmorrhage.

(8) Ulceration of the larynx, trachea and bronchi. In rare instances ulcers in these localities and adjacent structures by erosion of an artery may cause copious and fatal hæmorrhage.

(9) Morbid growths. Cancer, hydatids, actinomycosis are often attended with hæmorrhage from the lungs.

(10) Gangrene of the lung.

II. Sources of hæmorrhage situated outside the respiratory tract.

(1) Diseases of the heart. Valvular disease, especially mitral in advanced stages, may cause intense pulmonary congestion and hæmoptysis. Usually the hæmorrhage is mild.

(2) Aneurysm of the arch of the aorta or of branches of the pulmonary artery. In this instance there may be an oozing, but if beyond that the hæmorrhage is generally copious and rapidly fatal.

(3) Vicarious hæmorrhage. During menstruation this may occasionally be observed when there is amenorrhœa.

(4) Certain blood and infectious diseases. Purpura hæmorrhagica, scurvy, hæmophilia, anæmia and malignant types of acute infections. In these affections the hæmorrhage is due to the altered condition of the blood itself, and to the changes in the walls of the vessels.

(5) Arthritic endarteritis. Degeneration of the walls of the blood-vessels from arterio-sclerotic changes may cause hæmorrhage in those of advanced years.

Morbid Anatomy.—In a certain proportion of cases the seat of lesion is in the mucous membrane of the capillary bronchi, the vessels of which are ruptured from over-distention or from changes in the pulmonary parenchyma. In other instances the lesion consists of ruptures of the walls of larger vessels, which give way under ulceration and erosion. Excessive hæmorrhage from the lungs is generally of this nature.

On examination under the microscope the appearances are variable. Sometimes it is difficult or quite impossible to detect the point from which the hæmorrhage arose. If a considerable time has elapsed between the hæmorrhage and the examination the mucous membrane may be pale and exsanguinated. Frequently, however, it is swollen, with a tendency to bleed easily, softened and relaxed, of a bright red or dark color with points of ecchymoses.

The alveoli and bronchial tubes in various localities of the lung may contain blood clots, in some cases at a distance from the seat of hæmorrhage. When the blood comes from the walls of a cavity the latter will be found to contain a coagulum.

The lung tissue proper may present a paler appearance than normal.

It was formerly maintained that inflammation of the lungs was induced by the presence of decomposing coagula in the bronchi. This, however, does not appear to happen very often. In tuberculosis, on the other hand, infection may be very widely diffused throughout the lung by hæmorrhage.

Symptoms.—Generally hæmoptysis comes on without warning. Sometimes the patient first experiences a saltish taste, then expectorates the blood. Coughing is often present, and may precede the bleeding or be induced by it. In mild cases an ounce or more of blood may be lost and the bleeding cease. Again, small quantities of blood may continue to be raised at varying intervals for several days or more. In some cases the blood pours out of the mouth in a gush.

The accompanying symptoms as far as the hæmorrhage is concerned, it is needless to say, vary according to the amount of blood lost. In the milder forms the patient may experience no after-effects, except those of apprehension, which may induce palpitation and other nervous phenomena.

If hæmoptysis is excessive the symptoms are the same as those which follow excessive bleeding from other organs, namely, feeble pulse, faintness, prostration, tinnitus aurium, gasping respiration, etc. The presence of the blood in the bronchi adds greatly to the difficulty in breathing. After the bleeding has subsided, the sputum, especially in cases of pulmonary tuberculosis, often continues blood streaked for several days.

When a large vessel ruptures the blood pours out from the mouth, there may or there may not be a few attempts at coughing, and the patient expires. Fatal pulmonary hæmorrhage into a large cavity in the lung without hæmoptysis may occur in advanced forms of pulmonary tuberculosis.

The blood from the lungs is generally frothy, bright red, mixed with mucus, alkaline in reaction, and when coagulation takes place there may be air-bubbles in the mass. Casts in blood moulds may occasionally be seen. In some instances the blood may come up in the form of coagula.

Except in the milder varieties the temperature may rise from one degree to three or four, and occasionally more. The pulse is accelerated and excitable.

Not infrequently some of the blood is swallowed, and in consequence the stools are black. Again, the swallowed blood may cause nausea and vomiting and complicate the diagnosis by the vomiting of blood. Patients can often tell whether the blood comes from the respiratory tract or is vomited from the stomach. When coughing is present this is self-evident, but when the blood pours forth in large quantities it is not always so.

Hæmoptysis has no definite duration, it often ceases spontaneously. Sudden attacks without the slightest premonition may not be followed by any further symptoms for several months, and in some cases never at all. On the other hand, the first attack may be the beginning of a series, especially in the instance of pulmonary tuberculosis. In these cases there may be considerable intervals between the attacks.

Cough is frequently an important aggravating symptom, especially in tuberculous cases. It tends to prolong the hæmorrhage and induce its recurrence.

In affections of the aortic and mitral valve, especially mitral stenosis, hæmorrhages from the bronchial mucous membrane are not rare. They may be of a varying degree of severity.

Mild hæmoptysis may occur with aortic aneurysm. It may arise from either fungoid granulations on the mucous membrane, the result of perverted nutrition from pressure, or from exudation of blood through the walls of the sac. Excessive hæmorrhage from aneurysm arises from rupture of the sac and is rapidly fatal.

The so-called arthritic hæmoptysis is associated with gout and arterio-sclerotic change in the walls of the blood-vessels. These bleedings may occur at intervals for years and then cease. Chronic bronchitis is always present in these conditions. Emphysema is also often observed. It is probable that the hæmorrhage under these conditions arises from ulcers in the walls of the vessels.

Hæmoptysis appearing vicariously in place of the menstrual function may continue for some time without untoward results; on the other hand, it has proved to be the precursor of tuberculosis.

Slight hæmorrhages from the bronchial tubes in hysterical and delicate women sometimes occur. They are accompanied by cough and the blood comes from the upper portion of the respiratory tract.

Physical Signs.—Examination of the chest during an atttack of hæmoptysis should never be made unless auscultation is excepted, and this should only be practiced when it can be accomplished without disturbing the patient. Ordinarily physical signs do not reveal anything more than bubbling râles caused by the presence of blood in the bronchial tubes. Signs of prëexisting disease, on the other hand, may be very marked.

In some instances, after mild hæmorrhage, it may be difficult or impossible to locate definitely any change in the lung.

Diagnosis.—The fact that hæmoptysis is a symptom indicates that a determination of its cause and its relation to the morbid condition which induces it is, as mentioned, most essential. Hæmorrhage from the pharynx, œsophagus and stomach must not be confused with hæmoptysis. In epistaxis the blood may flow directly into the naso-pharynx, excite cough and be expectorated. Examination of the nose and pharynx will reveal the condition. Blood may come from the gums or pharynx. If from the first its source will be usually easily ascertained by examination of the mouth. If from the latter it is mixed with mucus and scanty. Hæmorrhage from the stomach or hæmatemesis may be distinguished from hæmoptysis by the following features:

Hæmoptysis.	Hæmatemesis.
History of cough and other symptoms pointing to pulmonary disease often present.	History of gastric disturbance and other symptoms pointing to disease of the stomach often present.
Sensation of weight and distress in the chest often present.	Epigastric distress and nausea precedes the symptoms.
The blood is raised by coughing, although it may be swallowed and vomited.	The blood is ejected by vomiting.
It is bright red, frothy, with small coagula mixed with mucus in small quantities, if at all, and alkaline in reaction.	It is clotted, coagulated, dark or has the appearance of coffee-grounds, mixed with mucus and food and acid in reaction.

Anæmic and delicate young women may sometimes raise blood in the morning. It comes from the gums, which are pale and spongy, and may run from the mouth at night and stain the pillows in the morning.

Prognosis.—As far as the attack is concerned the outlook is generally good. Exceptions to this favorable view occur in advanced forms of pulmonary tuberculosis and aneurysm when hæmorrhage may terminate the case.

It is well to recall in this connection that cases of pulmonary hæmorrhage which once evince a tendency to hæmorrhage often continue to be attended with hæmoptysis at intervals during the progress of the disease. Death takes place in fatal cases either from the loss of blood or from inundation of the lungs.

Treatment.—In the treatment of hæmoptysis a knowledge of the condition of the pulmonary circulation and the primary lesion is very important. It should be remembered that the morbid anatomical condition in pulmonary hæmorrhage is either hyperæmia of the bronchial mucosa and lung parenchyma, or rupture of an artery of the lungs.

When the blood comes up in small quantities, and the hæmoptysis continues for some days, the source of the bleeding is usually diapedesis from hyperæmia. When in large quantities it is probable that it comes from rupture of the walls of a blood-vessel, and if in very large quantities it is certain that such is the case. The general indications of treatment are lowering of the blood-pressure and reduction of the heart-beat.

In all cases the patient should be placed in the recumbent posture with the head and shoulders elevated. Apprehension should be allayed by assurances. Talking should be avoided as much as possible. The temperature of the room should be cool. Heat should be applied to the feet. Ice-bags may be applied to the chest, except in arthritic cases, when it is contra-indicated. The patient should eat small pieces of ice, holding the same in the mouth for some moments. The Chapman hot spinal bag applied to the middle and upper dorsal vertebræ may be used with decided advantage.* The diet should be simple and nourishing. Alcohol is not indicated. Water should be taken in limited quantities, as it tends to increase blood-pressure. Expectoration should be encouraged up to a certain degree, and the patient instructed to lie upon one side and not to change the position to the other side or back in order to clear the bronchial tubes more easily.

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* Digest of External Therapeutics, E. G. Rankin, M.D., 3d ed., 1904, Boericke & Runyon, N. Y., p. 440.

In some instances of hæmorrhage due to pulmonary congestion the amount of blood is small, and, beyond rest, little treatment is necessary. In many of these cases the slight loss of blood is often rather beneficial than otherwise. If free, on the other hand, interference is demanded. The bowels should not be allowed to become constipated. In case of moderate but persistent bleeding due to congestion, purgatives constitute one of the most effective means of lowering blood-pressure and may be administered with good effect. In fact, they will often succeed in this condition when other remedies fail. Sulphate of magnesia in half-ounce doses or calomel and jalap in ten-grain doses may be used with beneficial results. The writer has succeeded with the latter in sthenic cases of hæmorrhage of congestive origin after the failure of many other remedies. Dry cupping over the chest, hot foot-baths, leeches to the anus, temporary ligature of the extremities, in order to retard the return flow of blood to the heart, are other expediences which are recommended.

Styptics, chief among which are tannic and gallic acid which may be given in five-grain doses every two to every four hours, are of service in prolonged and persistent cases. Cough which tends to greatly aggravate hæmorrage may be allayed by heroin, codein or morphia.

In cases of profuse and alarming hæmorrhage, such as may occur in advanced stages of tuberculosis, remedies should be administered hypodermically, for no time can be lost. Morphia, millefolium, ergotole and atropine are to be employed under these circumstances.

Remedies with special indications are as follows:

Aconitum, when there is bright red, frothy blood, excitement, fear, high arterial tension, warm sensation in chest, flushed face and hacking cough, especially in the early stages.

Veratrum viride, in conditions of excessive pulmonary congestion with high fever, bounding pulse and rapid, short respiration, with absence of nervous excitement.

Hamamelis, in venous hæmorrhage, constant, but small in quantity, dark in color and coming without effort in coughing.

Geranium maculatum, hæmorrhages of bright red blood in tuberculosis, especially in the earlier stages, when aconite does not quickly relieve. Ten to twenty drops of the tincture may be given every fifteen to twenty minutes.

Millefolium, often of decided benefit in cases of pulmonary tuberculosis with cavities, and profuse hæmorrhage of red blood with little cough. Five drops of the tincture may be advantageously combined with a quarter of a grain of morphia sulphate and administered hypodermically.

Hydrastini hydrochloras has been highly praised by several writers for hæmorrhage from any part of the body. It is especially indicated in old subjects with bronchial catarrh and friable mucosa. It may be given in the second decimal trituration or hypodermically in quarter of a grain doses.

Ferrum phos., in anæmic persons who flush at the least emotion and in young growing persons with bronchial catarrh, headache and nose-bleed. The bleeding in these cases is slight and the blood bright red. There may also be œdema of the ankles, pains through the chest and shoulders, and undigested stools.

Ipecacuanha, bright red blood, cough, bubbling râles, nausea and vomiting.

Thlaspi bursa, in doses of thirty drops of the tincture hourly during the acute stage of the attack, recommended by Dr. H. P. Deady. As the condition improves the dose should be reduced to ten drops hourly on the second day, and finally ten drops three times a day and so continued for at least three to five days after the sputum is entirely free of blood. A special indication is bright red blood without the mental symptoms calling for aconite.

Erigeron, also recommended by Dr. H. P. Deady. More useful when the blood is bright red and the hææorrhage profuse, especially in the secondary break-up of old fibroid cases.

Phosphorus, in low forms, inflammatory conditions following hæmoptysis, especially in tall, slender persons inclined to stoop, and of tubercular diathesis. Additional indications are tightness of the chest, weakness and empty sensation of the abdomen, and profuse hæmorrhages which cease for a time and then return.

Acidum sulphuricum, in persistent cases, dark blood in small quantities, a constant oozing rather than much of a flow, with extreme weakness. It acts best in feeble anæmic and aged persons. The dilute acid or aromatic sulphuric acid may be used in full doses.

Morphiæ sulphas, in cases where there is persistent irritating cough, nervous excitement and quickened heart action, may be administered hypodermically with decided benefit in from one-eighth to one-third of a grain.

Gelatine has proved of service in hæmorrhage from the lungs when given hypodermically. This method of administration, however, is objectionable on account of the pain and the liability to sloughing and sepsis. Given by rectal injection these unfavorable features are avoided. The following is the method of procedure: Add 50 grammes of gelatine to 1 ¾ litres of boiled water. Boil the solution gently for one hour, evaporating to 1 litre, cool to the temperature of the blood and inject into the rectum a quarter of a litre slowly. Inject three times a day.

When the hæmorrhage is due to organic disease of the heart, the following:

Digitalis, when there is obstruction of the pulmonary circulation, pulse slow, irregular with cyanosis and difficult, sighing respiration.

Caffein, arsenicum and cactus grandiflorus may also be given according to their special indications.

PULMONARY INFARCTION.

SYNONYMS.—*Circumscribed Pulmonary Apoplexy, Hæmorrhagic Infarct of the Lungs.*

Pulmonary infarction is a circumscribed area of lung tissue which has become infiltrated with blood and which subsequently undergoes necrotic changes.

Ætiology.—The lesion arises either from emboli blocking branches of the pulmonary artery, or from thrombi forming in the same. The causes, therefore, may be accordingly classified under two heads, viz.:

I. The first or embolic group includes all coagula formed in any part of the venous systemic circulation and right side of the heart, and those solid bodies which force their way into the channels of the circulation.

A frequent source of pulmonary emboli is thrombosis of the veins of the leg. This is especially liable to occur in protracted fevers with pronounced anæmia and in traumatism resulting in laceration and inflammation of the veins. Thrombosis in the right side of the heart is also a frequent cause of embolism, especially the globular thrombi which form in the right auricular appendix.

Infarction may also follow childbirth and operation upon the pelvic organs in women, when veins in proximity of the arteries have been wounded. Embolism, however, following operative procedure is very rare.

Neoplasms, notably carcinomata and sarcomata, may grow into the circulatory channels. Small particles may become detached and pass into the circulation. These bodies to all intents act as emboli and may lodge in the lungs and give rise to infarction.

Infectious emboli are followed by abscesses. *Vide* Abscess of the Lungs.

Infarctions due to embolism of the pulmonary veins may occur, but are rare.

II. The second or thrombotic group include all conditions which favor thrombosis of the circulatory channels of the lung itself. Here disease of the heart, aorta and lungs are presumed to preëxist and act as casual factors, notably endocarditis, myocarditis, fibrosis, arterio-sclerosis and atheroma of the aorta. In these conditions the pulmonary circulation is feeble and the infarction is frequently the result of stasis.

Of all causes embolism is the most frequent, hence the greater proportion of cases fall under the first group of causes.

Morbid Anatomy.—A pulmonary infarction consists of a firm wedge-shaped body situated in the lung substance, usually in the lower posterior portion, near the periphery with its base or broad end toward the pleura. Sometimes it is sphenoidal in shape. There are usually several of these bodies which vary in size from a peanut to an orange. Their most frequent seat is the posterior portion of the lower lobe of the right lung. Sometimes they may occupy the greater portion of the lobe. When recently formed they are dark in color, and on section are hard, airless, projecting above the surface and presenting the appearance of an ordinary blood clot. Microscopical examination shows the presence of leucocytes and red blood-corpuscles in the alveolar cells and their septa, and sometimes hyaline infiltration of the small blood-vessels.

The pleura beyond the affected area is thickened, inflamed and covered with a fibrinous exudate. If the disease continues sufficiently long there is atrophy of the involved area, which becomes changed into a fibrinous mass.

If the circulation of the lungs is normal, very frequently the disturbance caused by an embolus is rapidly compensated. The formation of the infarction generally follows a prëexisting state of passive congestion. This occurs most frequently with mitral disease. Under ordinarily normal conditions the blood-pressure is sufficient to restore the circulation in the adjacent capillaries, but, when the venous pressure is increased, stasis, diapedesis and hæmorrhage follows the lodgment of the infarct.

In some instances pulmonary infarctions are pale and anæmic in consequence of extreme weakness of the circulation.

While every case of pulmonary infarction is due to embolus or to thrombus, every pulmonary embolus or thrombus does not give rise to infarction. This may occur when the embolus or thrombus is very small or very large. In the first instance the vessel obstructed may be too diminutive to be attended with extensive change, and the infarct is removed after liquefaction and granular degeneration of the blood clot. In the latter instance a main branch of the pulmonary artery may be occluded and instant death follow. There is in consequence no time for the formation of infarction in the lungs. (*Vide* Embolus and Thrombus of the Pulmonary Artery.)

Embolism of the lungs without infarction may also occur in the form of fat emboli from fractures with disorganization of the marrow.

Symptoms.—The symptoms vary according to the size of the infarction and extent of the lung involved. They are, however, not very distinctive. The patient is usually seized with pain in the side with dyspnœa of varying degrees of intensity, accompanied by mental distress, and followed in some instances by syncope. Hæmoptysis is frequent. Pure blood may be expectorated when the embolus from the peripheral circulation has lodged in a primarily healthy lung. A single hawking movement usually may be sufficient to expel it. Coin-shape masses of blood are then seen in the sputum. Sometimes, on the other hand, the amount of blood raised is copious. Again, it may be mixed with the bronchial secretion and assume a dark red gelatinous appearance. Hæmoptysis does not, as a rule, make its appearance at the onset of pulmonary infarction, but usually from eight to twenty hours after.

Cough is not essentially a symptom of infarction, when present it is often due to the accompanying heart lesions. It at first may not be affected by the formation of the infarction. But later it may increase, especially in the instance of infectious emboli.

Fever may be present from the beginning, or entirely absent throughout the course of the process.

Pleurisy of the fibrinous variety is probably the only complication in simple infarction.

Physical Signs.—If the infarction is sufficiently near the surface there is dulness on percussion and increase of vocal fremitus, and on auscultation occasionally bronchial respiration and usually crepitant râles. These signs are generally found in the right lower and middle lobe.

Diagnosis.—The presence of a sudden attack of severe pain in the chest with dyspnœa and hæmoptysis in conditions where thrombosis is known to exist, or when any of the causes likely to produce it are present, warrants the assumption of pulmonary infarction.

Errors in diagnosis are not likely to occur, although some writers mention broncho- and croupous pneumonia as possible sources of error. In the former the onset of the symptoms are more gradual and hæmoptysis not a marked symptom. The fever is also higher and of longer duration. In croupous pneumonia the physical signs and greater intensity of fever are distinctive.

Prognosis.—The outlook depends upon the condition of the lungs, the size of the embolus or thrombus and the presence or absence of pathologic bacteria. Cardiac or prëexisting pulmonary disease with consequent tendency to congestion render the prognosis much less favorable. Large or multiple infarctions are very grave.

In simple cases when the obstruction is small, the lung otherwise healthy and infection absent, the prognosis is good, for under these conditions the tissues are able to regain their vitality and absorption and resolution follow. A slight degree of pigmentation and induration may remain. When large infarctions tend to necrosis the future course depends upon whether or not bacteria find an entrance. If not, reactionary inflammation with connective tissue formation may occur, and the destroyed tissue be replaced by a cicatrix with probable puckering of the overlying pleura. If bacterial infection occurs abscess and gangrene may develop.

Treatment.—When thrombosis is suspected or known to exist every precaution should be exercised to prevent the detachment of an embolus, and as the greater number of cases arise from thrombosis of the peripheral veins, and most frequently those of the leg, the presence of the thrombotic process is often discernible. Phlebitis, swelling of the leg with hardness of the femoral vein, circumscribed swellings of the calf of the leg, or thigh, following protracted acute fever, sprains or injuries of any kind, are warnings that the sources of embolic infarction are at hand. In all these cases rest in bed should be rigidly enforced, with the application of cold compresses and internal administration of pulsatilla or hamamelis.

Massage is decidedly contra-indicated, as it tends to favor detachment of coagula. The diet should be liberal and nutritious, but bland and easily digested. The general line of the treatment, in short, should be supporting and strengthening. The bowels should be kept open and undue pressure on the abdomen avoided.

When the infarct has formed, if the pain and distress is severe, morphine in one-third to one-half grain doses should be given. Some prefer its combination with atropine. It should never, however, be administered for either the cough or hæmoptysis alone. If dyspnœa is excessive oxygen inhalation should be used. Should valvular disease or aneurysm of the aorta be present, digitalis or glonoine will probably be indicated as the condition progresses.

PULMONARY EMPHYSEMA.

Pulmonary emphysema is a condition of the lungs in which there is more or less permanent dilatation in the infundibular passages and alveoli with atrophy of the alveolar wall and loss of the normal elasticity of the lung tissue. There are several forms, namely, hypertrophic emphysema, compensatory emphysema and senile emphysema.

There are two other varieties which do not come under the above definition, but which for the sake of convenience are usually discussed under this head; these are acute vesicular and interstitial emphysema.

Hypertrophic Emphysema.

This is the form which is implied when the term emphysema or pulmonary emphysema is employed without other designation. It is also known as substantive, large lunged and idiopathic emphysema. It is characterized by enlargement of the lungs with distention of the alveoli, atrophy of their walls, dyspnœa, the clinical manifestations of defective aëration of the blood, and increase in size of the chest.

Ætiology.—The chief factor in the ætiology of emphysema is loss of elasticity of the alveolar wall from abnormally and persistently high air-pressure within the alveoli acting upon a weakened condition of their structures. The principal theories which have been set forth as explanatory of these changes are as follows:

The Inspiratory Theory.—This maintains that the air vesicles may become over-distended during inspiration in consequence of diminution of the expiratory power from loss of the normal elasticity of the alveolar walls, and that the latter condition is brought about by obstruction of the bronchi from chronic inflammation, whereby areas of collapse result and compensatory distention in adjacent lobules takes place. The same line of reasoning obtains in adhesions, collapse of the lung and consolidation, whereby the air vesicles in certain obstructed portions become over-distended and lose their contractile power.

The inspiratory theory was advocated by Laennec, and undoubtedly the phenomena attributed to it occur in the compensatory or vicarious form of emphysema, but it does not afford satisfactory explanation for the variety under discussion.

The Expiratory Theory.—This was supported by Jenner and is more widely accepted. It attributes the changes to over-distention of the unsupported portion of the lungs, such as the apices and margins, from violent expiratory effort when there is obstruction from any cause, such as excessive bronchial secretion, closure of the epiglottis in the act of coughing, straining, lifting or playing wind instruments.

The Nutrition Change Theory.—Various observers have given different explanations which may be included under this head. Cohnheim and others deemed it probable that there was a condition of congenital weakness due to defective development of the yellow elastic fibres in the lung tissue. This would obviously tend to result in over-distention on exertion, especially when of an unusual character. Brown-Sequard regarded the fundamental cause as a neurosis involving the terminal filaments of the pneumogastric nerve. Niemeyer attributed the changes to the result of violent expiration, as in coughing or straining, which forced the air upward into the upper and unprotected portions of the lungs and thus caused distention. Walshe was of the opinion that there were primary nutrition changes in the walls of the vesicles which were the result of inaction of adjacent portions of the lungs. Other theories are those of Villemin, who regarded hypertrophy of the vesicular septa with increased capacity as the cause, and who also suggested trophic neurosis of the terminal nerve filaments causing relaxation of the air vesicles.

Emphysema occurs at any period of life, even among children. It is more frequent among men than among women, from the fact that the former are more exposed to its causal influences. Hereditary tendencies seem to be factors in a large proportion of cases. According to Dr. James Jackson, of Boston, in eighteen out of twenty-eight cases one or both parents were affected. Persons with flat, undeveloped chests are predisposed.

The active causes in the ætiology of pulmonary emphysema are those which give rise to obstruction to the outlet of air from the bronchial tubes. The most frequent of these is chronic bronchitis, especially of the dry type. Here the coughing, as well as the bronchial changes, contributes to the condition. Whooping-cough may act in the same way. Asthma is a common cause and old asthmatics are frequently emphysemic. Certain occupations favor the development of emphysema, notably, playing on wind instruments, glass-blowing, lifting, straining, athletic exercise, etc.

Cardiac disease when it has reached the stage in which it causes pulmonary congestion frequently leads to changes in the lungs of an emphysematous nature.

Morbid Anatomy.—The cartilages are more or less calcified. On opening the thorax the lungs show great distention, overlapping the præcordium and sometimes bulging out of the incision. The unsupported portions, namely, the apices and margins, show a great degree of distention, especially if the condition is of long duration. The lungs are enlarged, pale and show loss of elasticity, and do not collapse either while *in situ* or when placed on the table. Instead of the moist crepitation normally present they impart a sensation to touch suggestive of parchment. The pleura is pale, with frequently absence of pigment in patches, termed by Virchow albinism of the lung. Beneath its surface enlarged air vesicles may be seen. These vary in size from one-half to three millimetres. Irregular bullæ sometimes the size of a bird's egg may project from the margins. These are the result of coalescence of the lobules.

Section shows that many of the contiguous lobules have been thrown together, their septa entirely obliterated or remaining as atrophied threads or bands. Although these distended lobules are more abundant on the anterior margin of the lung, they also appear at the inner surface of the lobe near the root where, in exceptional instances, vesicles as large as an egg may be found. The obliteration of the septa is the result of the pressure of the air which causes thinning of their walls and atrophy.

Microscopic examination shows that the earliest anatomical changes consist in enlargement and division of the nuclei in the walls of the air-cells as the result of distention. The cell division and proliferation continue and go on to disintegration and absorption. The atrophic process continues until the areolar walls are absorbed and the neighboring air-cells coalesce. In this change the capillary vessels disappear before the walls are entirely atrophied. A notable part of this process is the loss of the elastic tissue fibre. In protracted cases interstitial fibroid changes take place around the involved lobules, especially in emphysema which arises independently of bronchitis. The walls of the acini are very thin, the septa obliterated or marked by mere threads of fibrous tissue or fragmentary lines, the remnants of the capillaries.

The epithelia of the air-cells undergo fatty degeneration, while a pavement layer remains in the large distended air spaces. In consequence of these changes it is apparent that the vascular area for aërating the blood is diminished, and that the elasticity of the lung tissues which materially assists in respiration is greatly lessened.

The bronchial tubes exhibit changes such as occur in chronic bronchitis and bronchiectasis. The large bronchi show roughening and thickening, while often submucous tissue appears above the surface in folds. In the small bronchi, in cases of long standing, dilatations occur especially when there is peri-bronchial fibrosis.

A most important change is dilatation and hypertrophy of the right side of the heart, which arises as the consequence of the extra work required of the organ in order to propel the blood through the diminished vascular area of the lungs. The tricuspid orifice is enlarged and the valve cusps thickened at their margins. In advanced cases especially the hypertrophy of the heart may be general. The changes in the heart may not be observed until after death, because of the overlapping of the over-distended and voluminous lungs. Dilatation and atheroma of the pulmonary artery and its branches may also be found, and chronic interstitial nephritis and nutmeg liver are not uncommon.

Symptoms.—Emphysema is essentially an affection of middle and advanced life, although in a limited number of instances it occurs in childhood. It is often fairly well advanced before it gives evidences of its presence. The symptoms in a great degree depend upon the presence of complications, especially that of failure of cardiac compensation.

Dyspnœa is the most persistent and characteristic symptom. In the earlier periods of the affection it may only appear on slight exertion or during attacks of bronchitis, or it may be aggravated by the latter. As the affection advances it becomes more or less constant being greatly increased by exertion or distention of the stomach. Respiration is frequently wheezing, prolonged and harsh.

Cyanosis is a pronounced symptom. A characteristic feature is the comparative comfort and the moderate degree of activity which the patient enjoys notwithstanding its presence. In other affections of the lungs, or in cardiac disease in which there is interference with the proper aëration of the lungs sufficient to produce such a degree of cyanosis, the patient would be in bed in a position of orthopnœa.

Cough is common; it is usually loud, hoarse and wheezy. It generally occurs in paroxysms and is aggravated by cold or damp weather.

Expectoration will be profuse if bronchitis is present; otherwise it is scanty, being raised in small so-called "pearly" masses.

Bronchitis is common and frequently the cause of aggravation of symptoms. In some instances it may induce paroxysms of asthma. As the case advances the bronchial symptoms become more persistent, especially in cold weather, so that they become inseparably cöexistent with the emphysema. Amelioration is marked in mild and warm weather.

Hæmoptysis is an occasional and rare symptom. It may occur from rupture of an atheromatous artery. The amount of blood is usually small, though it may be excessive and cause death.

The pulse is small and indicates that the arteries are poorly filled. Gastro-intestinal disturbances in the form of dyspepsia, malassimilation, flatulent distention of the stomach and bowels and constipation may arise from obstruction of the portal circulation. Headache and drowsiness and vertigo after exertion from the undulations of the cerebral circulation are common. Clubbing of the fingers and toes sometimes occurs. The physiognomy may show changes. The face may be full, lips thick, mucous membranes congested, and in advanced cases there may be a general cyanotic appearance. In elderly persons, when the condition has existed a long period, the position and conformation of the thorax are highly characteristic—the shoulders are rounded, the chest barrel-shaped and thin, yet somewhat muscular.

The course of the affection is slowly progressive. Usually several years elapse before characteristic symptoms manifest themselves—in very rare instances a few months. Frequent attacks of bronchitis favor its development.

The complications that commonly arise are dilatation of the heart with the attending phenomenon of failing compensation. In addition to bronchitis acute intercurrent affections may occur, especially in the form of pneumonia.

PHYSICAL SIGNS.—*Inspection* shows antero-posterior enlargement of the chest, which is aptly described as barrel-shaped. The ribs and sternum are prominently elevated, causing apparent shortening of the neck. The lower portion of the thorax appears enlarged and the intercostal spaces widened, especially in the hypochondriac regions. The sternal fossa is deepened, the clavicles everted, the curve of the spine much increased and the back rounded. A zone of dilated cutaneous veins may be seen along the line of attachment of the diaphragm. This, however, is not peculiar to emphysema alone. Inspiration is short and quick and the distention of the chest slight. During coughing a bulging outward of the supraclavicular spaces may occur. This is sometimes called the emphysematous tumor. It arises partly from the distention of the emphysematous apices and partly from distention of the jugular vein and sinus. *Mensuration* shows the actual enlargement of the chest due to increase in the antero-posterior diameter and loss of expansion on inspiration. The chest does not expand, but is elevated on inspiration, the respiratory movement, while apparently energetic, exerts little influence in causing expansion. There may be some distention in the upper abdominal region during inspiration and occasionally a transverse curve at the level of the twelfth rib across the abdomen. There is usually loss of the apex-beat and marked pulsation in the epigastrium. Distention and pulsation of the veins of the neck may be regarded as evidence of dilatation of the right side of the heart.

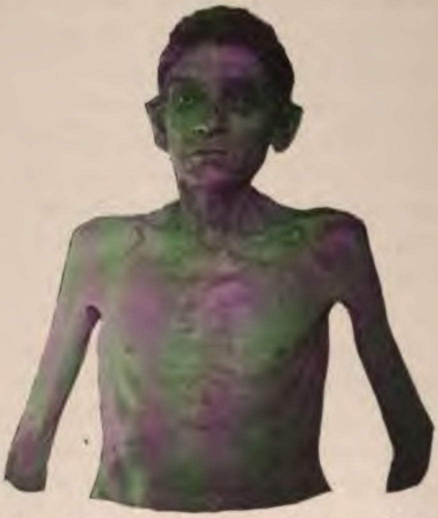

FIG. 40.—Barrel-shaped chest in pulmonary emphysema. Metropolitan Hospital.

Palpation shows decrease of vocal fremitus, the apex impulse being feeble or imperceptible. There is, on the other hand, a decided impulse in the epigastrium. The whole thorax will be felt to move *en masse*.

Percussion gives exaggeration of resonance. The note is frequently drum-like or tympanitic, especially over the apices. The normal area of cardiac dulness may be obliterated from the lungs overlapping the heart. The upper margin of the liver may also suffer encroachment for the same reason, the inferior border of the lungs being from one to two inches below normal. The area of resonance may be extended beyond the normal posteriorly, at the level of the first or second lumbar vertebra; splenic dulness may likewise be diminished.

Auscultation.—The inspiratory sounds are shortened and enfeebled. If there is much bronchitis, râles, both sonorous and sibilant, will be audible. Vocal resonance may be diminished or interrupted. The most characteristic feature is the prolongation of the expiratory murmurs. The normal relation of inspiration to expiration of one to four is reversed to about four to one.

The normal vesicular murmurs undergo changes, being harsher and rougher. Changes also indicative of fibrosis, bronchiectasis and atelectasis may be present.

Expiration is often harsh and of low pitch and the heart-sounds indistinct, except in the epigastrium where they may be clear. In advanced conditions there is a tricuspid regurgitation and accentuation of the pulmonic sound.

Diagnosis.—The physical signs, the history of previous attacks of bronchitis, dyspnœa which, although marked, permits of considerable exertion and prolongation of expiration, are the main features which usually render the recognition of emphysema easy.

There are really no conditions which can be mistaken for emphysema. Pleurisy with effusion, fibroid pneumonia and pneumothorax present on superficial examination a certain degree of resemblance, but the physical signs distinctive of these conditions on closer examination will remove all sources of error.

Hypertrophy of one lung and hypertrophy of the lungs in athletes may also present the appearance of emphysema. Both these conditions are very rare, and in both there is normal percussion and auscultation. The general physical condition of the patient is also normal, while the contrary holds in emphysema.

Prognosis.—The danger in emphysema lies not in the disease itself, but in the complications which it is prone to induce. As these are of a grave nature it follows that the presence of emphysema must be regarded with concern. Although the disease is progressive, the patient may live for years. Much depends on the exciting cause. The prevalence of bronchitis must be taken into consideration. If persistent, it lends an unfavorable aspect. Broncho-pneumonia, pneumonia, asthma and sudden heart weakness are all possible complications. Of especial importance is the presence of cardiac lesions; when these are present the liability of failure of compensation should always be taken into account when making a prognosis.

Treatment.—The degenerative changes which characterize emphysema when once established are beyond the reach of medicinal agents. The aim of treatment, therefore, should be the arrest of further progress of the process, the avoidance of complications and the relief of symptoms.

Each attack of bronchitis aggravates the primary condition and renders the patient more susceptible to subsequent attacks, while pneumonia in emphysematous subjects is especially fatal. Every effort should be made, therefore, to protect the patient against these complications. He should be warmly clothed, wearing woolen undergarments, and should take exercise in moderation, carefully avoiding fatigue, lifting, straining, and everything that tends to cause dyspnœa. Whenever it is possible, a dry, equable climate, free from dust and wind, should be chosen for residence. When this is not possible the patient should remain in-doors when the weather is damp or foggy or the wind high. The diet should be carefully regulated. In the earlier periods of the disease it should be most nutritious, comprising articles of food rich in nutrition. In the advanced conditions, as the digestion is generally impaired, it should consist of the most easily digested articles of food, amylaceous and saccharine articles being avoided. In some cases a milk diet proves suitable. Tobacco and alcohol, as a rule, should be forbidden. As the venous engorgement commonly present to a greater or less degree tends to gastro-intestinal disorders, it is very important that the bowels should be kept open.

In children with asthma which develops emphysema the antrum and the nasal passages should be examined for the presence of disease.

Compressed-air baths are often effective and some good results have been obtained by their systematic use. Inhalations of oxygen are also of service.

The administration of medicine is limited to the treatment of the complications and attendant conditions, and as these are chiefly bronchitis, asthma, bronchiectasis and dilatation of the heart, in addition to the following, reference should be made to the treatment of those subjects.

The most important remedies are as follows:

Antimonium arsenicosum, of great value, especially in advanced conditions with excessive dyspnœa, severe paroxysmal cough, asthma and general debility.

Antimonium tartaratum, for the moist cough with much mucus.

Arsenicum iodatum, in cases where there are evidences of arteriosclerosis, senile decay and weak heart.

Calcarea carbonica, for chronic bronchitis, especially in fleshy persons who perspire freely and in women with excessive menstrual flow.

Ipecacuanha is often of great service when there is bronchitis with dyspnœa, wheezing, with great weight and anxiety about the præcordium, and violent cough with nausea.

Quebracho, especially for the relief of the dyspnœa.

Calcarea phosphatica, useful in elderly persons with arterio-sclerosis and arcus senilis.

Phosphorus is also useful in cases with arterio-sclerosis and those subject to fatty degeneration.

Aurum may prove of service in subjects with nervous symptoms, arterio-sclerosis and urine of low specific gravity.

Veratrum viride is recommended by some observers in conditions of excessive dyspnœa with extreme engorgement of the venous circulation. One to three drops of the tincture every half hour for three or four doses are said to prove effective in affording relief.

Strychnia is one of the best remedies when the heart is weak; one-twentieth to one-tenth of a grain may be given in divided doses during twenty-four hours. It acts not only as a cardiac stimulant and tonic, but often also as an expectorant by strengthening the walls of the bronchi. It may be continued for some time. Spartein and agaracine have been recommended for the same purpose.

For the gastro-intestinal disturbances, lycopodium, nux vomica and carbo vegetabelis will prove of service.

When there is high arterial tension, glonoin 2x dilution in one-drop doses is especially indicated. Additional remedies for this condition are amyl nitrite, sodium nitrite and potassium and strontium iodide.

Other remedies that should be considered in emphysema are arsenicum, belladonna, chininum arsenicosum, lachesis, lobelia, and acidum nitricum.

Compensatory Emphysema.

This term signifies an emphysematous condition in one portion of the lung which arises as the result of disease in some other portion, and which by a vicarious increase in the volume of the air-cells compensates for the loss caused by the latter. The usual causes are fibroid induration of the lung, pulmonary tuberculosis, pleurisy with adhesions, especially when the latter are situated at the inferior border of the lung and pneumothorax. The most marked examples are found with fibroid induration and in the neighborhood of tuberculous areas and old cicatrices.

In the early periods of the affection the distention is physiological, the alveolar walls being simply stretched and not atrophied, but later these undergo degenerative changes and atrophy. The walls of the contiguous cells thereupon coalesce and a true emphysema results.

There are no symptoms of special significance. In some instances there may be physical signs, but these are not generally reliable. The condition may be inferred when the morbid states which favor its development exist.

Atrophic or Senile Emphysema.

This is essentially a senile atrophy of the lungs. It is generally observed in old persons who give the impression of being withered. Sir William Jenner called the condition "small lunged" emphysema.

The pathological changes consist in atrophy of the vesicular walls with consequent coalescence of the adjacent air-cells and weakness incident to age is often an underlying cause. The tendency to pulmonary inflammations in valvular disease, especially mitral stenosis, even in those not advanced in years, is also another important predisposing influence.

Primary catarrhal pneumonia usually occurs in young children. The causal factors are essentially the same as those of the croupous variety, and the micrococcus lanceolatus is often associated with it.

Secondary catarrhal pneumonia arises frequently as a complication of acute infections, notably measles, which furnishes a large percentage of cases. It is also frequent in influenza, scarlet fever, diphtheria, and less frequent in typhoid fever, variola and erysipelas. With influenza it is quite common among adults. In these affections the toxine of the specific infection is doubtless the direct cause of the disease. Its occurrence under these conditions is very common, especially with measles, where it is responsible for a large number of deaths in children under five years of age. In fact, broncho-pneumonia stands next to intestinal disease in the mortality of early childhood. It is most fatal during the first two years of life. In adults, secondary broncho-pneumonia is not so frequent. In typhoid fever, according to some observers, the lobar variety is more common. In the aged it is not infrequent in association with gout, emphysema, debility and valvular diseases of the heart.

Acute simple bronchitis, especially if it is seated in the smaller bronchi, may extend to the capillary tubes and air-cells and then set up broncho-pneumonia. Such cases are common and in many instances they might be considered in one sense of the word primary, their origin being simply by extension of the inflammatory process which involves the bronchi.

BACTERIOLOGY.—It is assumed that the specific and direct cause is the action of bacteria on the mucous membrane of the smaller bronchi. But inasmuch as the disease is not uniform in its manifestations, that is, it is either primary, or a secondary inflammation arising in connection with some acute infection, such as measles, influenza or diphtheria, it would appear that infection may take its origin from several forms of micro-organisms. Bacteriological examination shows the presence of the micrococcus lanceolatus, the streptococcus pyogenes either alone or with the former, the staphy-

In rare instances the pleura may rupture, causing pneumothorax with or without pleurisy.

Symptoms.—Urgent dyspnœa suddenly appearing after violent coughing or other possible causes, especially in childhood, points to its presence and constitutes the most prominent symptom. If pneumothorax occurs the dyspnœa is excessive.

Treatment.—Absolute quiet and prevention of coughing should be attempted. The cough should be treated by the indicated remedy and inhalations of steam. The strength and heart should be supported by stimulants.

CATARRHAL PNEUMONIA.

SYNONYMS.—*Broncho-Pneumonia, Lobular Pneumonia, Disseminated Pneumonia, Capillary Bronchitis.*

Catarrhal pneumonia is an inflammation of the terminal bronchi and air vesicles which constitute the lobules of the lung.

The inflamed areas occur in isolated patches made up of the involved lobules or group of lobules. There is always bronchitis of the capillary and larger bronchial tubes.

It is no longer regarded as possible for the terminal bronchi to be the seat of acute catarrhal inflammation without involvement of the lobular structures. Hence the existence of capillary bronchitis as a distinct and separate disease is not now recognized by the majority of the latest writers. It is true some continue to make the distinction, but it is noticeable that none give a definite differential clinical diagnosis between the two lesions.

Ætiology.—*Predisposing Influences.*—Exposure, climatic changes and bad hygienic surroundings, especially impure air, are important predisposing influences. It thus naturally follows that the disease is more prevalent in the winter and early spring and among the poor. A long continued recumbent position also predisposes. The influence of age is very decided. Childhood and old age favor its development to a very great degree; the disease, however, belongs more to childhood. Children suffering from malnutrition, rickets or diarrhœal diseases, are very liable to broncho-pneumonia. In advanced life the presence of debility, chronic valvular disease of the heart, emphysema and gout are strong predisposing factors. The smooth, airless, grayish in color and somewhat raised above the surface.

Emphysema is frequently found on the surface of the lung, along its anterior border and surrounding the inflamed lobules.

The morbid processes which characterize broncho-pneumonia show decided variability in their distribution and nature. In some instances the principal changes are chiefly situated in the bronchioles, there being little definite pneumonic consolidations. This type corresponds to "capillary bronchitis." Yet microscopical examination reveals many of the air-cells and adjacent bronchioles filled with the products of inflammation. In others there are disseminated areas of peri-bronchial consolidations with patches of collapse with a considerable portion of the lung remaining crepitant. This is the most common type. Another form is that when the consolidations appear to coalesce at certain points, and the greater portion of the lobe becomes consolidated. The consolidation is not uniform, but is interspersed with areas of dark congested tissues. This variety is called the pseudo-lobular.

An essential feature of catarrhal pneumonia is its tendency to spread from one part of the lung and to involve additional areas.

Aspiration pneumonia and hypostatic pneumonia classified by some writers as forms of broncho-pneumonia are discussed separately in this work.

Microscopical examination shows interstitial inflammation of the bronchi and alveolar walls as the special feature of broncho-pneumonia. The calibre of the involved bronchus is found to be plugged with a mass of exudation composed of swollen epithelia and leucocytes. A sectional view of the tube in its long axis may reveal irregular dilatations. Its wall and those of the air-cell connected with it are œdematous and infiltrated with leucocytes. The alveolar septa are infiltrated with round cells and the blood-vessels are surrounded with emigrated leucocytes.

The air-cells are also filled with leucocytes, swollen epithelia and mucus, those near the periphery show much less exudate. Hæmorrhagic and purulent exudation may be observed in some cases; the former is not common, the latter occurs when streptococci and staphylococci are present. Fibrinous exudations may also be found in some cases.

The collapsed areas show quite a degree of congestion, and the cavities of the alveoli are obliterated by the falling in of the walls or by the exudation which is more or less hæmorrhagic.

There is generally decided leucocytosis of the polynuclear variety. Absence of increase of the leucocytes is of grave significance as it implies feeble resistive power. A decided leucocytosis, on the other hand, does not necessarily signify a favorable outlook.

The pathogenesis of bronchi-pneumonia may be summarized as follows: The inflammation involves at first the bronchioles and with more or less rapidity it extends to the communicating air-cells, or involves the surrounding alveoli by contiguity, in both instances a patch of catarrhal pneumonia results. Less often the affected bronchioles become plugged by mucus or by œdema of the walls and collapse of the lobule follows, followed in turn, it may be, by pneumonic consolidation resulting from the entrance of micro-organism or from extension of inflammation from adjacent structures.

Symptoms.—The symptoms of catarrhal pneumonia display a decided variability in their manifestations. This doubtless accounts for the conflicting statements of various writers when discussing the disease. In many instances the onset is associated with an acute bronchial catarrh which passes to the capillary bronchi and air-cells. In others the bronchial catarrh may be slight and the initial symptoms like those of croupous pneumonia with all its suddeness. Between these two types there may be many gradations and deviations.

In primary catarrhal pneumonia the invasion of the disease is often sudden and attended with a chill or rigor. The temperature rises rapidly, ranging up to 104° F., but is variable. In children the pulmonary process may be overlooked on account of the prominence of other symptoms. In adults the onset may strongly resemble simple bronchitis of a severe type.

Secondary catarrhal pneumonia is the variety usually observed. It is commonly preceded by bronchitis of the larger tubes, but its symptoms are often more or less obscured by those of the primary disease. Its appearance during the course of some primary disease is attended with decided increase of temperature and rate of respiration. In some cases there are rigors or chills.

In catarrhal pneumonia of both primary and secondary origin the characteristic symptoms are increase of the rate of respiration which may run as high as eighty per minute, a variable and irregularly remittant temperature arises ranging from 101° to 104° F., sometimes making its appearance gradually, rapid pulse, and cough at first dry and harassing and later attended with expectoration of glairy muco-purulent sputum, sometimes blood streaked. The expectoration is usually not copious. Pleuritic pains may be present, but are not so often observed as in the croupous variety. Dyspnœa is prominent and frequently the patient cannot lie down. Cyanosis appears as the process advances, the countenance appears dusky, lips and finger-nails blue. The skin is moist and clammy.

A feature of the temperature of catarrhal pneumonia is that, ordinarily, it rises more gradually than that of croupous variety and when defervescence begins it does not, with some exceptions, reach normal, short of three or four days. During the course of the disease there will be sudden rises occurring irregularly, this denotes as a rule the invasion of new areas of the lung.

While the above is an outline of the symptoms of more or less characteristic cases, there are variations from this type which call for especial consideration. Catarrhal pneumonia, it should be remembered, is commonly observed in childhood and advanced life, but more especially in the former. In children the disease may be ushered in with convulsions and the cerebral symptoms be so marked that the pulmonary are overlooked. In these cases in addition to the convulsions there may be great restlessness, delirium alternating with stupor, high fever and marked prostration. After persisting from two to five days cerebral symptoms subside and the pulmonary appear.

Sometimes the attack may simulate croupous pneumonia with its abrupt onset, chills or convulsions and rapid rise of temperature, reaching 104° to 105° F. and falling by crisis so that it is only by the subsequent course of the disease, if at all, that a distinction can be made. In some instances again the child may seem ill with cough and coated tongue, loss of appetite and occasional vomiting and fever. An examination may at first fail to reveal pneumonic consolidation, yet a day or two later this condition will be recognized either at the extreme base, axilla or near the root of the

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course in broncho-pneumonia than in the croupous variety. The exudates undergo a fatty change, become lighter in color and are finally absorbed or expectorated. At the same time the round cell infiltration disappears from the septa and the tissues gradually return to normal.

When the course of the disease does not tend to resolution the following conditions may result:

(1) Tuberculosis: In those cases of broncho-pneumonia where the process is situated in the apex, especially in children with measles, when the pulmonary complications have persisted for weeks, caseation of the lung may result. This may occur because the tubercular infection was present *ab initio* or because the lung became infected subsequently. Many cases, however, which have been regarded as simple at first with apparent subsequent infection are tuberculous from the beginning.

(2) Suppuration and Gangrene: These processes may result in cases where the micro-organisms are especially virulent. Fetid pultaceous masses surrounded by congested and inflammatory œdema are present.

(3) Fibroid Degeneration: In other instances when the process towards resolution is slow, fibrosis of the connecting tissues of the septa and more or less overgrowth of the same within the alveoli develops. These changes result in fibroid hardening of the lung tissues. It is rare in simple broncho-pneumonia, but comparatively common in that associated with tubercular infection.

The morbid changes which are most liable to be associated with broncho-pneumonia are pleurisy and a general toxic infection. Pleurisy is less frequent than in croupous pneumonia, but the areas of broncho-pneumonic inflammation when near the surface are often covered with a pleural exudate. In children purulent infection after pneumonia is more frequent than in adults, and in many of these instances the antecedent pneumonic process is catarrhal or broncho-pneumonic.

PHYSICAL SIGNS.—*Inspection* shows the increased rate of respiration and in advanced conditions cyanosis. In very grave cases there may be retraction of the base of the sternum and lower costal cartilages during inspiration, indicating defective lung expansion.

lung where the air does not enter so well. In such cases the fever may run for a week or two, when the temperature falls to normal. The symptoms of this type are sometimes described as subacute.

In children, also, it should be remembered that usually there no sputum is raised. It is swallowed. There are also gastro-enteric symptoms in the form of vomiting and occasional diarrhœa. The pulse and respiration become extremely rapid; restlessness and prostration are marked. In catarrhal pneumonia associated with measles the fever is usually high.

In elderly persons with debility or some cardiac weakness or lesion catarrhal pneumonia often comes on very insidiously. In these cases respirations may become very rapid, sometimes reaching sixty per minute, or they may be very much less. The temperature usually runs a low curve, ranging from 99° to 102° or 103° F. A low temperature and a gradual onset is usual in all cachectic and asthenia catarrhal pneumonias. In fatal cases the fever may be absent entirely, or may even be subnormal; a low or subnormal temperature may be observed toward the end. In unfavorable conditions, in all types of the disease as the process advances, the dyspnœa becomes more intense, the cyanosis more pronounced, the sensibilities gradually obtunded by the deficient aëration, and the patient sinks into unconsciousness and dies from pulmonary œdema or heart failure from over-distention of the right ventricle.

General catarrhal pneumonia is another type. It develops suddenly with chills followed by high temperature, marked prostration, head, chest and loin pains, rapid respiration, rapid pulse quickly becoming very feeble, cough at first dry and later attended with blood streaked and muco-purulent sputum, cyanosis and delirium.

The course of the catarrhal pneumonia like its symptoms is marked with great variation. If extensive it may end fatally in a few days, in less severe cases it may run a comparatively mild course for several days or a week, then suddenly the temperature rises from perhaps 100° to 104° F. or more, and the patient rapidly passes to a dangerous condition and dies. Ordinarily the course of the disease is not less than a week or more than three weeks. Some persist for much longer. Thus the course is not definite.

When resolution is once established it pursues a more rapid

Catarrhal Pneumonia.	Lobar Pneumonia.
Onset usually gradual, often secondary to bronchitis and acute infections.	Onset abrupt, usually primary.
Fever irregular, usually runs at a lower range and declines by lysis after a varying duration.	Fever high and so persists, falls between the fifth and tenth days, often by crisis.
Sputum, tenacious, glairy and in adults often blood tinged, not very copious. Dyspnœa marked, respiration often very rapid with pronounced cyanosis.	Sputum rusty, often copious. Dyspnœa usually not marked, breathing rapid and panting, but cyanosis slight.
Physical signs show general bronchitis, areas of consolidation, the former predominating, and subcrepitant râles.	Physical signs show crepitant râles in the first stage with dulness, followed in the second stage by the evidences of consolidation which predominate over all other signs.
Process usually bilateral.	Process usually unilateral.
Duration indefinite.	Duration definite.
Bacteria mixed, streptococci in the majority of cases, diplococcus pneumonia in many.	Diplococcus pneumonia in 90 per cent. of cases.

In children with cerebral symptoms it is often impossible at the onset to determine the true condition, but a close watch should be kept upon the lungs. If the disease is pulmonary the signs of the catarrhal pneumonic inflammatory process will develop within a few days.

In primary catarrhal pneumonia which by aggregation of the inflamed lobules involves a considerable portion of one lobe, especially when the onset is sudden with chill, followed by high fever, it is almost impossible to differentiate the condition from lobar pneumonia. Osler gives as a point of difference that in catarrhal pneumonia a slight lesion is more likely to be found on the other side. This, however, is only suggestive.

Acute broncho-pneumonic tuberculosis in the early periods of the disease may closely simulate non-tubercular catarrhal pneumonia, especially when the latter is situated in the apex. The symptoms and signs may be almost identical. In doubtful cases examination of the sputum will establish the diagnosis. The presence or absence of elastic tissue fibres and above all the tubercle bacilli are the determining factors. In all cases of catarrhal pneumonia when resolution is delayed the sputum should be examined for tubercle bacilli.

Prognosis.—Catarrhal pneumonia must always be regarded as serious, but generally speaking, in previously healthy persons, es-

Palpation confirms the defective expansion and may show increased vocal fremitus, but in this respect is often negative, especially when the areas of consolidation are very small.

Percussion shows impaired resonance or positive dulness, usually over the base of the lungs, but frequently death ensues before consolidation can be definitely determined. Hyper-resonance may sometimes be observed if the areas of consolidation are small. Usually this note is of unequal quality in different parts of the base of the same lung. This may be considered a sign of value. Of important significance also is the change of note over the same area, that is, it is sometimes tympanitic and sometimes high pitched.

Auscultation reveals numerous fine subcrepitant and sibilant râles. When consolidation involves a large portion of the lung the respiration may be bronchial or tubular.

Auscultation is by far the most important factor in the physical signs, and in the early period of the disease the presence of fine subcrepitant râles may be the only sign, except the rapid breathing.

Diagnosis.—The presence of some prëexisting disease likely to induce catarrhal pneumonia, the physical signs of consolidated areas and general bronchitis, the latter predominating over the former, the presence of subcrepitant râles, high rate of respiration, marked dyspnœa, cyanosis, irregular and prolonged fever point to the disease. If all these symptoms are present, yet consolidation cannot be made out, the diagnosis should still be made, as in this instance the consolidated patches are too small to elicit dulness. The latter is the condition formerly called capillary bronchitis, which is not now, as stated, recognized as a separate disease entity.

There are two diseases with which catarrhal pneumonia may sometimes be confused, these are lobar pneumonia and acute pulmonary tuberculosis. The distinction in regard to the former, except when the consolidation is extensive, and in certain types in children as mentioned, may be determined by the following :

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Prognosis.—Catarrhal pneumonia must always be regarded as serious, but generally speaking, in previously healthy persons, especially if the disease is primary, the outlook is favorable. In the debilitated and enfeebled it is the reverse. The extent of the lung involved is a very important element, for the greater it is the more serious the symptoms and the more unfavorable the prognosis. Rises in temperature and increased rate of the pulse are of unfavorable significance as they indicate involvement of additional areas of the lung. This tendency of the disease process to spread and extend should always be taken into consideration.

With the acute infections catarrhal pneumonia is always a grave complication, especially in whooping-cough, scarlet fever and influenza—perhaps less so in measles, although, as mentioned, its mortality when associated with measles is large. Notwithstanding this unfavorable outlook many desperate cases recover. When the disease occurs in children under two years the prognosis must always be regarded with deep concern, as at this age the mortality is large. If the child is weak or rachitic it is usually fatal. It is this type of constitution, it should be recalled, which are most prone to the disease.

In advanced life catarrhal pneumonia is usually associated with debility or with some other prëexisting morbid state. Under these circumstances the prognosis should be guarded. Many of such cases die. The presence of cardiac lesions lends a peculiarly unfavorable aspect to the course of the disease.

Treatment.—The room of the patient should be as large as circumstances permit and well ventilated. The temperature should be kept at 68° F. and the atmosphere moistened by steam vapor. The patient should remain absolutely quiet, and while the dorsal decubitus should be maintained the postureshould be frequently changed, as thereby it is thought tendencies to collapse of the lobules are avoided. At the beginning of the attack it is advisable to relieve the bowels with castor oil, or small repeated doses of mercurius dulcis. The diet should be light and nutritious, consisting of milk, koumyss, broths and egg albumen. The latter may be made palatable by mixing it with water and sweetening. If milk disagrees, as it often does, it may be mixed with lime water or "modified." In some instances it disagrees in any form and the diet must be confined to broths and egg albumen.

It is customary to apply a cotton-batting jacket. This is to be commended. Poultices are not to be employed except in the instance of pleuritic complications, when they may be applied over the seat of pain. A small flaxseed meal poultice may be slipped under the cotton jacket.

Bathing in the form of sponging with warm or tepid water should be employed throughout the course of the disease. It relieves irritability and tends to reduce the temperature. The water should be lukewarm. In case the temperature runs high and internal remedies and sponging do not reduce it, a wet pack may be employed with good results, beginning with a temperature sufficiently warm to be agreeable and reducing gradually to 80° F. When there are cerebral symptoms either with or without high temperature, the wet pack or cool sponging often prove of great benefit. Some physicians prefer a bath.

Ice applications have been highly indorsed, especially by the Germans and by some English observers. It is claimed that they keep down the inflammatory process and relieve the pleuritic pains. The ice should be applied in soft rubber bags and the surface of the skin protected by a layer of flannel.

Dyspnœa is a condition which often calls for especial attention. In milder forms relief may be obtained to a certain degree by gently massaging the chest with warm olive oil or vaseline mixed with spirits of turpentine. In severe cases the bronchial tubes fill with mucus, the skin becomes cyanosed and respiratory failure threatens. This is more likely to occur in those cases in which bronchitic symptoms predominate. There is not only obstruction in the smaller tubes, but the excessive accumulation leads to collapse of the lobules. In this emergency active measures are indicated to arouse the patient and to excite the expulsion of the mucus. Alternate douches of hot and cold water projected on the nuchæ have proved of value. Hyperdermic injections of ether may also be tried.

Inhalations of oxygen are advisable and should be employed with the onset of cyanosis. The heart, it is needless to emphasize, requires careful watching and any tendency to failure should be met with brandy or whiskey and strychnine.

Aconitum.—When there is arterial excitement, high fever and restlessness this is the first remedy in the early stages.

respiration being diaphragmatic. Many of such patients live for an indefinite period, manifesting nothing more than somewhat labored respiration, but if subjected to great physical effort they suffer from very urgent dyspnœa and should they become victims of broncho-pneumonia death usually results. The physical signs in such cases are those of emphysematous areas in association with compression. There will also be evidences of enlargement of the right ventricle with dilatation.

Prognosis.—In congenital pulmonary collapse the severity of the case will be indicated by the degree of cyanosis. In cases where it is very marked, death usually takes place a few days after birth. In other instances the child may live several weeks or longer and finally succumb.

In non-congenital cases, if the area of the lung involved is limited, it is not usually serious, but when extensive it is most always fatal. The fact that the affection, grave in itself, is usually engrafted upon a primary disease, also grave, such as broncho-pneumonia, sufficiently explains the condition. It is very fatal when arising as a complication with whooping-cough. With pleurisy and pericardial effusion it is also very grave. In short, extensive collapse of the lungs always lends an exceeding unfavorable aspect to all conditions, both acute and chronic, with which it is associated.

Treatment.—In newly born children with collapse of the lungs, any mucus or meconium within reach, obstructing the air-passages, should be removed. Bathing at a temperature of 100° F. with thorough massage and sprinkling the chest with cold water may be employed with benefit in some instances. After removal from the bath the infant should be wrapped in cotton batting. Closing the infant's nose and blowing into the mouth is another measure to inflate the lungs.

In non-congenital cases the treatment varies according to the primary condition. In acute lesions liable to be attended with pulmonary collapse, or where from the nature of the case there is reason to apprehend it, the patient should be instructed to practice full inspirations at intervals. His position should be changed frequently. The application of cold water to the back of the neck, pouring it on in a stream, is a measure which has received indorsement as a preventive. It may also be employed as a remedial agent when the

with nutritious and strengthening diet, and constitutional and tonic remedies. In every instance the patient should be kept under supervision until all traces of the disease have disappeared and the lungs have entirely cleared.

ATELECTASIS.

SYNONYMS.—*Collapse of the Lungs, Apneumatosis.*

Atelectasis (ἀτελής, imperfect; ἔκτασις, expansion) is an absence or removal of air from the air-cells attended with collapse of their walls.

Ætiology.—It may be either congenital or acquired. In both instances it is a secondary condition.

The congenital variety is the most frequent. It arises from the imperfect establishment of the function of respiration in the new-born and may occur as the result of the following conditions: (1) Obstruction of the air-passage by mucus, meconium or enlarged bronchial glands. (2) Congenital weakness of the thorax. (3) Struma, rachitis or syphilis. (4) Defective action of the diaphragm. (5) Pressure on the brain from cerebral hæmorrhage whereby the lungs are prevented from expanding.

The acquired form occurs in a variety of conditions, viz.: (1) Obstruction by viscid mucus in bronchitis and bronchial pneumonia, especially in children with weak constitutions, whooping-cough and extension of diphtheria into the bronchi. The process consists of a more or less absolute occlusion of the smaller bronchi with mucus, pus and the products of inflammation. In the case of dipththeria the membrane is the obstructing body. (2) Direct pressure upon the lungs. This may occur from pressure of pleural effusions, hydrothorax, pneumothorax, pericardial effusion, aneurysm of the aorta and neoplasms, very extensive hypertrophy of the heart and an excessive degree of meteorism or ascites which presses upward on the diaphragm and compresses the lower portions of the lungs. (3) Conditions which impair the respiratory function, such as certain cerebral diseases, weakness of old age, or that of prolonged fevers, or great weakness from any cause. (4) Deformities of the chest. In extreme kyphoscoliosis the lung may be compressed and a condition of true collapse result, especially if the condition

respiration being diaphragmatic. Many of such patients live for an indefinite period, manifesting nothing more than somewhat labored respiration, but if subjected to great physical effort they suffer from very urgent dyspnœa and should they become victims of broncho-pneumonia death usually results. The physical signs in such cases are those of emphysematous areas in association with compression. There will also be evidences of enlargement of the right ventricle with dilatation.

Prognosis.—In congenital pulmonary collapse the severity of the case will be indicated by the degree of cyanosis. In cases where it is very marked, death usually takes place a few days after birth. In other instances the child may live several weeks or longer and finally succumb.

In non-congenital cases, if the area of the lung involved is limited, it is not usually serious, but when extensive it is most always fatal. The fact that the affection, grave in itself, is usually engrafted upon a primary disease, also grave, such as broncho-pneumonia, sufficiently explains the condition. It is very fatal when arising as a complication with whooping-cough. With pleurisy and pericardial effusion it is also very grave. In short, extensive collapse of the lungs always lends an exceeding unfavorable aspect to all conditions, both acute and chronic, with which it is associated.

Treatment.—In newly born children with collapse of the lungs, any mucus or meconium within reach, obstructing the air-passages, should be removed. Bathing at a temperature of 100° F. with thorough massage and sprinkling the chest with cold water may be employed with benefit in some instances. After removal from the bath the infant should be wrapped in cotton batting. Closing the infant's nose and blowing into the mouth is another measure to inflate the lungs.

In non-congenital cases the treatment varies according to the primary condition. In acute lesions liable to be attended with pulmonary collapse, or where from the nature of the case there is reason to apprehend it, the patient should be instructed to practice full inspirations at intervals. His position should be changed frequently. The application of cold water to the back of the neck, pouring it on in a stream, is a measure which has received indorsement as a preventive. It may also be employed as a remedial agent when the

In congenital pulmonary collapse the high pulmonary blood-pressure may cause the ductus arteriosus and the foramen ovale to remain patent. In non-congenital forms there may be dilatation or hypertrophy of the right ventricle.

Symptoms.—The symptoms in the new-born are rapid superficial breathing, a feeble whining cry, drowsiness, lividity, coldness of the extremities, muscular twitchings and convulsions. There may be congenital anomalies of the circulatory organs in association.

In non-congenital or acquired forms the symptoms appear secondarily to some primary condition and are clinically more or less obscured by the latter. In bronchitis and broncho-pneumonia, especially when they occur in feeble children and the aged, collapse of the lungs is most frequent, but if slight may not be observed. In cases where it is extensive the symptoms are grave. The respirations suddenly increase in rate, at the same time becoming labored, respiratory effort being carried on by the superior and anterior portions of the chest. The pulse becomes small, feeble and rapid and the surface cold, especially that of the extremities. The patient gradually becomes cyanosed and falls into unconsciousness.

PHYSICAL SIGNS.—*Inspection*, when a considerable portion of the lower lobe is involved posteriorly, as is frequently the case, discloses falling in during inspiration of the supra- and infra-clavicular spaces and of the lower portion of the chest. This is due partly to atmospheric pressure and partly to the action of the diaphragm. *Palpation* may be negative; in extensive cases fremitus is decreased.

Percussion shows dulness if the areas involved are extensive. Localized areas of emphysema may mark the situation when the collapsed areas are small and give localized semi-tympanitic resonance.

Auscultation shows in the part involved diminution or absence of the normal vesicular murmur which is displaced by bronchial breathing. The signs of the lesions with which atelectasis is associated are also to be taken into consideration, the most constant being the subcrepitant râles of broncho-pneumonia.

In aplasia of the lung with kyphoskoliosis, lung expansion, on account of the fixed twisted condition of the chest, is confined,

respiration being diaphragmatic. Many of such patients live for an indefinite period, manifesting nothing more than somewhat labored respiration, but if subjected to great physical effort they suffer from very urgent dyspnœa and should they become victims of broncho-pneumonia death usually results. The physical signs in such cases are those of emphysematous areas in association with compression. There will also be evidences of enlargement of the right ventricle with dilatation.

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condition already exists. Inhalation of oxygen and of compressed air are of service. Stimulants and stimulating nutrition are generally always indicated. Strychnine often proves useful.

CROUPOUS PNEUMONIA.

SYNONYMS.—*Pneumonitis, Lobar Pneumonia, Inflammation of the Lungs, Fibrinous Pneumonia, Lung Fever.*

Pneumonia is an acute infection associated with the diplococcus pneumoniæ or micrococus lanceolatus of Fränkel and characterized by inflammation of the lungs with croupous exudation into the air-cells and small bronchi, fever and termination by crisis from the fifth to the tenth day.

It is evident that what is now known as pneumonia was recognized by Hippocrates (B. C. 460–387) and others who described it as peri-pneumonia or pleuritis, but the ancient writers and those who followed up to the time of Laennec (1819) made no distinction between pleurisy and pneumonia. Laennec was the first to clearly differentiate between the two conditions, at the same time recognizing the three stages of the disease as now held. This great achievement of Laennec naturally paved the way for future additional advance in the pathology and diagnosis of pneumonia. Rokitansky in 1841 made known from a macroscopic point of view the nature of the exudate. Ziemssen in 1858 followed with valuable data in regard to the geographical distribution of the disease.

Ætiology.—Pneumonia is very generally prevalent and while not limited to any particular locality it is of more frequent occurrence in the temperate zone and in certain areas within the same. It has been stated that in this country it is more frequent in the Southern than in the Northern States, but, according to the Census Reports, there is not much difference. While climate in itself does not appear to exert any great amount of influence, season has a decided effect as the disease is more frequent in the winter and early spring.

Cold in itself is not conducive to pneumonia, and in northern cities where the winters are evenly cold the death-rate from pneumonia is low. It is the sudden changes in temperature from cold to comparative mildness and the reverse which appear to furnish the element of danger and which favors the development of the disease.

Pneumonia is not limited to any period of life, but there are certain ages which seem to favor its development. In the very young it is common. According to Holt's statistics,* of five hundred cases of pneumonia in children, 15 per cent. were in their first year; 62 per cent. from the second to the sixth year; 21 per cent. from the seventh to the eleventh, and 2 per cent. from the twelfth to the fourteenth. These figures show that the disease is more frequent up to the sixth year, when it declines. In adult life there does not appear any age in which it is more prevalent, but the question of age bears strongly on the prognosis, being more unfavorable after forty-five.

Pneumonia is more frequent among men than women, probably because the former are more exposed to its ætiological influences. Debilitated constitutions, cachexias, chronic diseases, such as Bright's, valvular disease of the heart, and notably alcoholism, are predisposing causes. Bad hygiene and a confined indoor life are also contributing ætiological influences. A strong constitution, on the other hand, lessens the susceptibility, although the robust often succumb. The disease is more prevalent in cities than in the country, as naturally follows from its infectious nature. Previous attacks undoubtedly predispose, as shown by pneumonia occurring several times in the same person.

Exposure, getting wet and chilled, and consequent "catching cold" has, since the recognition of pneumonia as a disease entity, been regarded as the active cause. While its influence in this respect is undoubted, it does not act in the way it was formerly thought. The modern view holds that the results of exposure by lowering the vitality and congesting the mucous membrane of the respiratory tract place the patient in a condition favorable for infection by the coccus. As the effects of exposure are more liable to be manifest after sudden atmospheric changes, it is doubtless for this reason, at least in part, that the disease is more prevalent in climates where these conditions are prone to occur.

Pneumonia sometimes appears endemically and epidemically. Evidences of the first characteristic are afforded by apparent sporadic outbreaks in certain localities, as, for example, in a building where a number of cases of severe type appears at the same time or in rapid succession. Such conditions may be attributed to bad hygiene which favors the propagation of the coccus and which places the patient in a condition of feeble resistance to its power.

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* L. Emmett Holt, Diseases of Infancy and Childhood, 2d edition, 1902, New York.

The epidemic character of the disease is manifested by its spread throughout a whole community, scattered cases occurring with undue frequency at certain times in a certain town or suburban locality. These features, together with its tendency to run a definite cycle, such as only observed in other infections, and the disparity which often exists between the local lesion and the grave constitutional symptoms, suggested the infectious nature of pneumonia long before the fact was clearly determined.

BACTERIOLOGY.—In 1872 von Jürgensen first suggested the infectious nature of pneumonia.

On April 30, 1881, Pasteur published an account of the discovery of a micro-organism in the saliva of a child dead from hydrophobia. He did not, however, recognize the fact that this was in any way associated with pneumonia. In September of the same year Sternberg inoculated rabbits with his own saliva and isolated a bacillus known as the "infection of sputum septicæmia." This organism was subsequently shown to be the same as that discovered by Pasteur. In 1883 Friedländer demonstrated the presence of a micro-organism in pneumonic sputum which is now called the pneumo-bacillus, an entirely different organism from that of Pasteur and Sternberg. In 1884 the coccus discovered by Pasteur was demonstrated by Fränkel to be intimately connected with the causation of pneumonic inflammation, and since that time it has been associated with the latter's name. There was considerable confusion at first in regard to the micro-organism of Fränkel and that of Friedländer.

The micrococcus lanceolatus, diplococcus pneumoniæ (Fränkel), or micrococcus pneumoniæ, commonly called the pneumococcus, although that term is also applied to the micro-organism discovered by Friedländer, is an oval or conical or somewhat lance-shaped organism occurring in pairs with the broad ends in apposition. It varies in size and those bodies making up the pairs are not always of the same size. Its presence is demonstrated in cover-glass preparations with the usual solutions and the Gram method. It is encapsulated when in the body, but not in the cultures outside the body. When stained by carbol-fushin the coccus becomes intensely red, while the capsule becomes light red. It thrives on agar and bouillon, but not on gelatin. Great variation is observed in its virulence. In some cases no effect will be produced by inoculation; in others a more or less extensive purulent exudation will appear at the point of inoculation. Injection into the lung itself causes pleurisy, pericarditis or a typical croupous pneumonia. Its virulence is diminished by cultivation.

This coccus is found in the exudation of the lungs in nearly all cases (about 90 per cent.) of genuine lobar or croupous pneumonia. It is also found in broncho-pneumonia, acute peri- and endocarditis, acute pleurisy, empyema, acute purulent meningitis and otitis media; less frequently in synovitis, peritonitis, osteomyelitis and abscess formations. In croupous and broncho-pneumonia it is present in large numbers. It is also occasionally found in the nose, Eustachian tubes, larynx and buccal secretions of healthy persons.

Weichselbaum in an examination of one hundred and twenty cases of pneumonia found the coccus of Fränkel, the streptococci pneumoniæ, a coccus similar to the former, but occurring frequently in chains, the bacillus pneumoniæ and the streptococcus.

The bacillus of Friedländer is an entirely different organism. It is a short, oval body inclosed in a capsule which usually contains one bacillus, rarely two or more. It is anaerobic and non-motile. It is decolorized by the Gram method and when treated with an aqueous staining solution the capsule becomes only slightly colored. The organism flourishes on agar and potato, but not so much on gelatin. It still remains uncertain whether the presence of the bacillus of Friedländer in the alveoli is of any ætiological importance.

The means by which the micrococcus lanceolatus enters the organism is probably by inhalation. Its first location is doubtless in the lungs, thence it is carried to non-distant parts of the circulation, such as the pleura, pericardium, endocardium, meninges, etc. The latter are only in rare instances invaded independently of the lungs. At first the disturbance caused by the coccus is local, but it is very speedily followed by general symptoms. Secondary infection from the streptococci, the staphylococci and other infectious micro-organisms may occur in various organs.

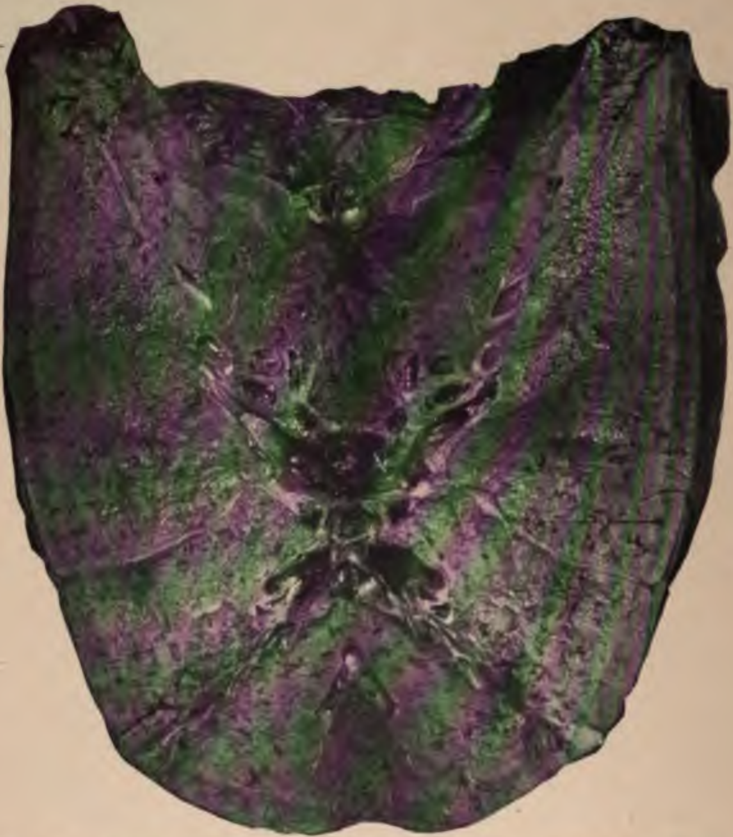

FIG. 41.—Croupous pneumonia. Longitudinal section of lung, the lung being cut partly through and the parts folded back. Upper portion in a condition of gray hepatization, lower, red hepatization.

Morbid Anatomy.—The pathological changes in pneumonia are positive and definite. They consist of an inflammation of the lung substance characterized by a croupous exudation into the air-cells. Since the time of Laennec three stages have been recognized, namely, engorgement, red hepatization and gray hepatization.

In the first stage the color of the lung tissue becomes a deeper red and its substance heavier and firmer. It pits on pressure, but crepitates, although less so than in health, and floats when thrown into water. The air-cells can be still inflated from the bronchus. On section bloody serum exudes on pressure. Microscopic examination shows the capillaries distended and tortuous, the alveoli more or less filled with swollen granular epithelia, detached alveolar epithelia and red and white blood-corpuscles. There is also œdema of the alveolar walls. Minute hæmorrhagic points are seen scattered through the interlobular and subpleural tissues.

The second stage, or that of red hepatization, is characterized by consolidation of the affected portion of the lung which is now firm, solid and non-crepitant, sinking when placed in water. The general appearance of the lung is red, but often it is mottled gray when an entire lobe is involved. It may appear enlarged, showing the indentations of the ribs. On section it will be seen that the cause of the consolidation is the presence of an exudation into the air-cells and finer bronchi, those up to one-fifth of an inch in diameter being filled, giving the lung the appearance of liver, hence the term hepatization. The quantity of the exudate is frequently large—sometimes as much as four to six pounds in weight. The red color is due to the intense engorgement of the blood-vessels and the number of red blood-corpuscles in the exudate. Further examination of the cut surface shows it to be coarsely granular. This is due to the masses of exudate protruding from the alveoli and infundibula which are rendered more prominent on account of the retraction of the lung substance after section. This feature shows with a varying degree of distinctness depending upon the size of the air-cells which vary according to age, being about 0.1 mm. in diameter in infants, 0.15 to 0.16 mm. in adults and 0.25 mm. in old age. On scraping the cut surface a reddish-brown liquid containing a mass of granulated bodies comes away. The latter are the fibrinous moulds or exudations which fill the alveoli.

Microscopic examination shows the exudation to be made up of coagulated fibrin containing in its meshes red blood-corpuscles, polynuclear leucocytes and alveolar epithelia. In some instances the red blood-cells greatly predominate, while in others the epithelia are found almost exclusively. Cover-glass preparations will demonstrate the presence of micro-organisms, as mentioned, viz., the micrococcus lanceolatus of Fränkel, the bacillus of Friedländer and in some cases the streptococcus and staphylococcus.

The third stage, that of gray hepatization, is distinguished in favorable cases by the commencement of resolution and in unfavorable by suppuration. At this period the color of the lung becomes gray. Its substance is still firm and liver-like, with increased weight. On section it is seen to be more moist than in the previous stage, the granules less distinct, appearing to have undergone liquefaction, and the tissues less friable. Microscopically the change in color is found to be due to the presence of leucocytes in the alveolar cells and the destruction of the red blood-corpuscles. At the end of the process the lung is filled with a yellowish fluid.

The duration of the several stages is difficult to determine. The first seldom continues after the second day; the second is probably present for from four to eight days; the third for a period varying from a few days to a week or more, depending upon the time occupied in the clearing up of the lung.

The several stages are not always clearly defined, but may more or less merge one into the other. The inflammatory process may involve any part of the lung, but in the greater proportion of cases it begins in the lower lobes posteriorly, more frequently on the right side. One or both lungs may be primarily affected, but generally only one. The process may sometimes begin in the central portion of the lungs and in rare instances in the apex.

Accompanying the pneumonic process there is always more or less bronchitis with the characteristic pathological changes in the bronchial mucosa. The bronchial glands are œdematous, congested, soft and pulpy. The pleura over the affected area is always involved when the process is near the surface and in many instances there is an exudation giving the membrane a slightly cloudy appearance. In some cases the exudation may consist of a creamy layer. There may also be some serous exudation.

Post-mortem examination of the heart shows the left side empty and the right side distended and filled with firm clots which, when withdrawn, come away as a cast. The blood in the pulmonary vessels in the side affected may show evidence of a thrombotic tendency. The lymphatics are filled with an exudate similar to that in the alveoli. A frequent phenomenon is the presence of the so-called fibrinous casts in the finer and terminal bronchi. These bodies, first discovered by Remak in 1845, consist of fibrin, pus-cells and a few cylindrical epithelia. They are more abundant between the fourth and seventh day, although they may occur later.

The characteristic change in the blood is leucocytosis which appears early, continues during the course of the disease, and disappears with the crisis. The leucocytes vary in number from 25,000 to 35,000 on an average, but the count may run as high as 64,000 per cubic centimeter. There is an increase of fibrin which becomes more marked as the disease advances, increasing from 4 to 1000 to as high as 10 to 1000. Coagulation is rapid.

Polynuclear corpuscles are abundant at first; later the ëosinophiles increase. The red blood-corpuscles are diminished, but undergo little change. Micro-organisms are not often found.

The presence of leucocytosis is of important significance. It first makes its appearance at the time of the chill and continues throughout the fastigium and falls with or just after the crisis. It remains in delayed resolution, purulent infiltration, empyema and gangrene. In mild infection the leucocytosis is slight; in severe it is decided. In some cases of severe infection it is slight or absent. These almost always die.

According to Müller the resolution of the fibrinous exudation is caused by the action of a ferment in the hepatized area induced by the large mass of leucocytes which migrate there between the stages of red and gray hepatization. The leucocytes cause an acid reaction which aids in the dissolving or digestion of the exudate.

The presence of the pneumococci in the endocardial inflammatory deposits is an important and interesting feature.

Implication of the cerebral meninges may also occur, giving rise to meningitis, the inflammation being usually cortical. The pneumococci are also present in this condition. The parotid glands may likewise show the pneumococci.

Examination of the kidneys discloses swelling of the parenchyma. The liver is enlarged, showing venous engorgement and cloudiness of the cells. The spleen is flabby and likewise enlarged. Croupous exudation may in rare instances be found in the colon, stomach and other localities.

Symptoms.—Pneumonia generally manifests itself with clearly-defined and characteristic symptoms, but in a certain number of cases it shows more or less marked deviation from the standard type.

Prodroma are not common, but they may appear in a small number of cases, consisting of malaise, slight bronchitis or "cold."

THE TYPICAL FORM.—The onset is sudden. There is a severe chill which may continue for ten or fifteen minutes or much longer. If the temperature should be taken during the chill it would be seen that the fever had already commenced. After the chill the reaction occurs. The patient complains of headache and general pains, the face becomes flushed, the respiration accelerated; there is a dry cough, sharp lacinating pain in the side and the pulse is full and quick. The temperature is from 103° to 104° F., but as a rule it does not reach its maximum until the second or third day. When seen at this time the condition of the patient is decidedly characteristic. He lies upon his back, the respiration is hurried and often accompanied by an expiratory grunt, while the alæ nasi may dilate with each inspiration. The cheeks are flushed, sometimes one more than the other and the expression is that of anxiety. At first the eyes are bright, but later they are dull. The cough is soon attended with expectoration which may be blood-tinged and very tenacious. Examination of the chest will reveal the physical signs of the disease. After persisting for from four to ten days, very rarely twelve, the temperature falls and the patient, after a period of great distress and suffering, passes into a condition of comparative comfort. Such in brief is an outline of the typical variety. In view of the important significance of the symptoms, their separate consideration is presented in detail as follows:

The Chill.—The special features of the chill are its suddenness and severity. In a small proportion of atypical cases it is imperfectly developed or absent, especially in the pneumonia of the aged, in alcoholics and in children. In rare instances there may be a series of chills, the symptoms of pneumonia not making their appearance after the first chill, but after one of the subsequent chills. It has been stated by some writers that this phenomenon occurs in patients with great myocardial weakness, but it occurs in others as well.

The Fever.—The temperature rises with great rapidity in most instances, 103° to 104° F. on the first day. On the second or third day it attains its acme and tends to remain at that point. There are, however, morning remissions, usually of about a degree. In some cases it may run from 102° to 103° F., while in others there may be hyperpyrexia. On the fourth or fifth day there is frequently a decided fall. This is called the false crisis and it may be looked upon as a favorable sign. The temperature, however, rises again and continues until the real crisis. The pseudo-crisis may sometimes occur on the third day, but this is not common. There may also be in the course of the disease a fall of temperature on the seventh, ninth and eleventh day, which is followed by a rise, but which affords a certain amount of relief to the fever.

In elderly persons, children and alcoholics the temperature may follow a much lower course. Instances of afebrile pneumonia are on record. An increase of fever during the course of the disease may be considered as due to additional absorption of toxic matters or of implication of a new area of the lung. If there is renewal oı the pain the latter is probable.

If the fever persists after the twelfth day, complications and sequelæ of a serious nature may be generally looked for, namely, purulent infiltration, empyema, typhoid conditions, abscess, gangrene and tuberculosis.

If the temperature falls to normal and then rises to 101° to 102° F., it may be due to rapid absorption of the toxic matter from the lungs. Just before death the temperature may fall rapidly or there may be an ante-mortem rise.

The Pulse.—During the chill the pulse is small, but when the fever appears it is full and bounding. In moderately severe active cases it is from one hundred to one hundred and sixteen. A pulse of one hundred and twenty-five to one hundred and thirty is a cause of anxiety. It is seldom dicrotic. Feebleness, irregularity and extreme rapidity are unfavorable indications. In some cases it

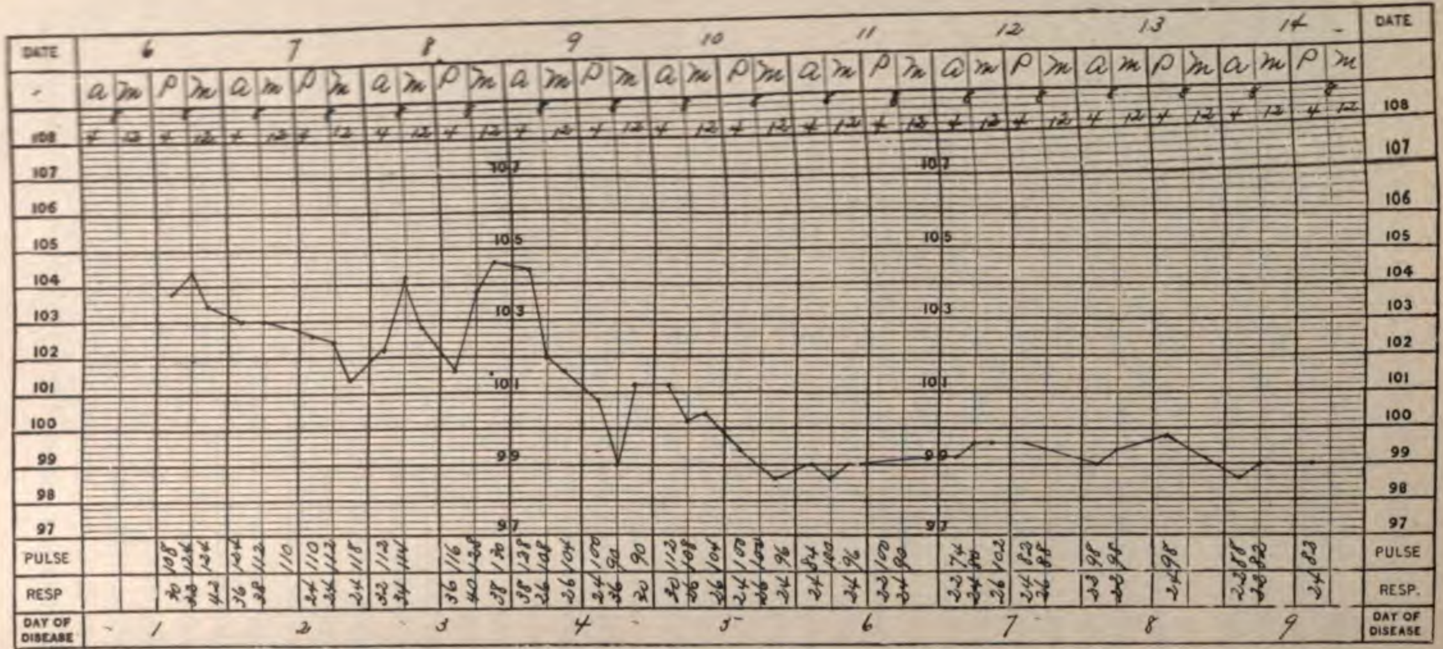

FIG. 42.—Temperature chart of a case of croupous pneumonia, showing fall of temperature by crisis on the fourth day. The case is interesting not only on account of the early crisis, but also from the fact that it illustrated the endemic type of the disease, being the third consecutive case of pneumonia in the patient's family. Patient, male, aged 39 years. Flower Hospital.

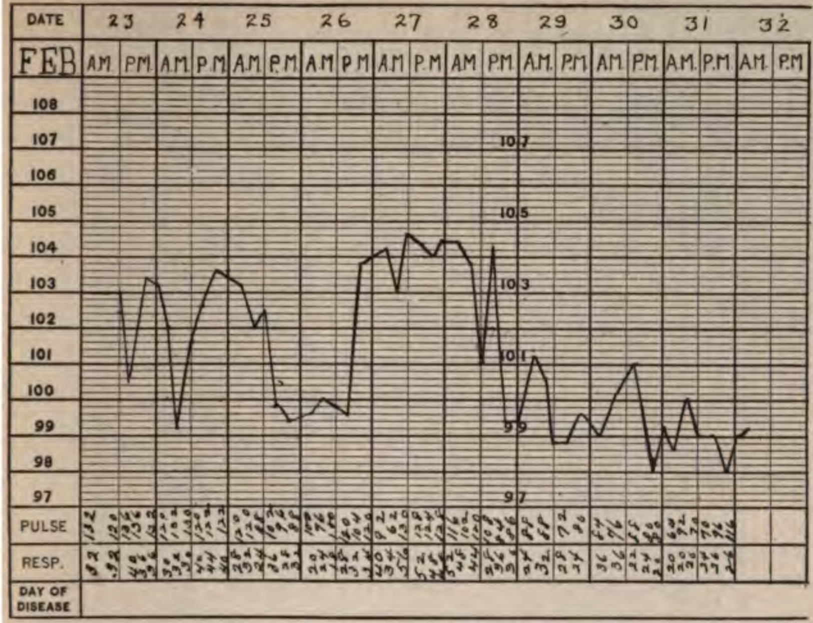

FIG. 43.—Temperature of a case of croupous pneumonia, showing exaggerated pseudo-crisis on the fifth day, actual crisis on the eighth. The initial chill occurred the day before the chart was commenced. Patient, male, aged 24 years. Flower Hospital.

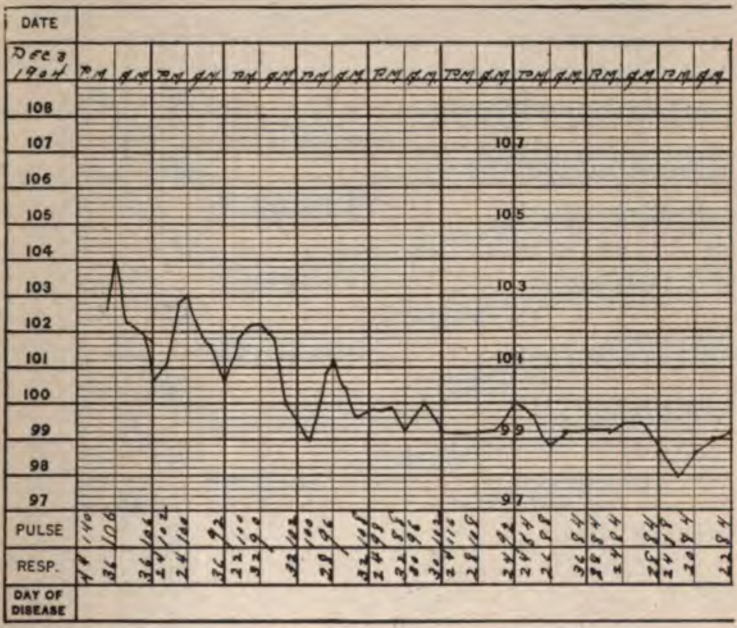

FIG. 44.—Temperature chart of a case of croupous pneumonia. Chart commences on the second day of illness. Pseudo-crisis on the third day with fall by lysis. Patient, a male, aged 44 years. Flower Hospital.

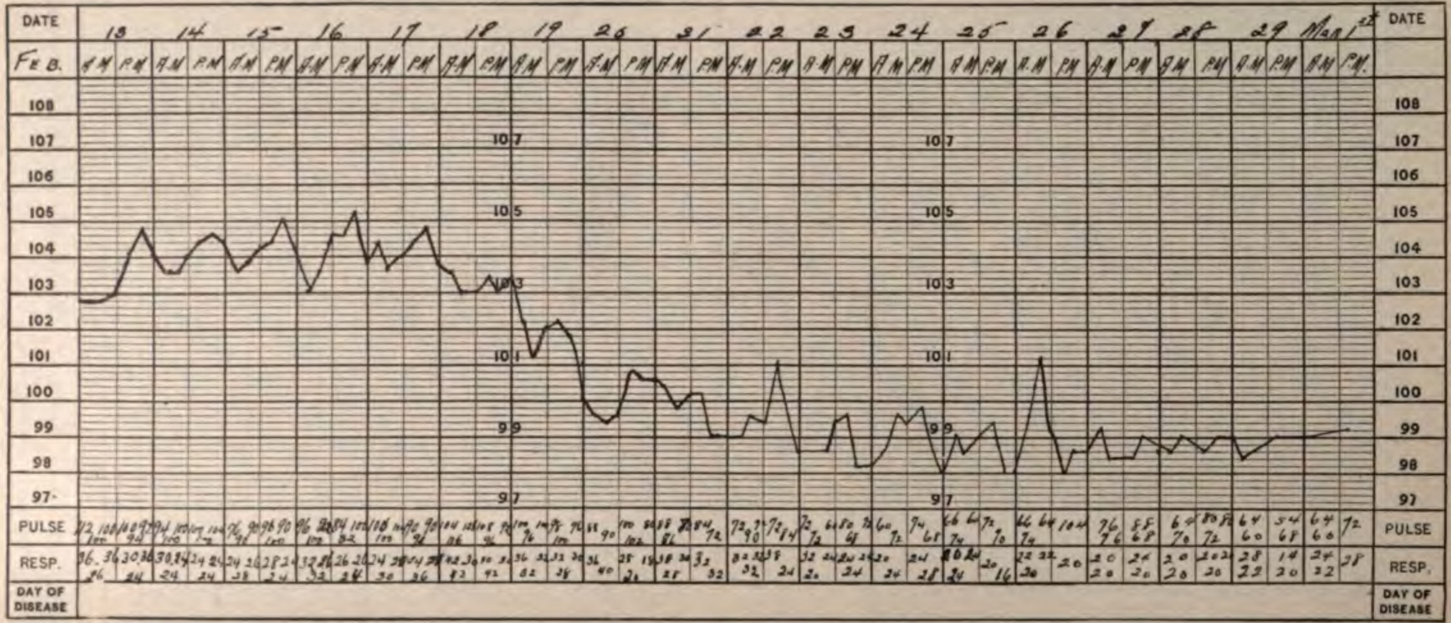

FIG. 45.—Temperature chart of a case of croupous pneumonia, showing fall of temperature by lysis, beginning on the eighth day. The initial chill occurred two days before admission to the hospital. Patient, male, aged 36 years. Flower Hospital.

may show no signs of weakness, especially in the strong and robust. While a most important element in prognosis the pulse alone is not reliable.

Respiration and Dyspnœa.—In active cases it is from thirty-five to forty-five per minute, with a pulse of one hundred and ten to one hundred and twenty. A rate of fifty is unfavorable, though by no means necessarily so. In asthenic cases the rate of respiration is less frequent than in the sthenic. In children it may be greatly accelerated, even eighty or more. The movements of the chest are superficial or limited. When there is pain the limitation may be mostly voluntary, that is, the respirations are restrained. Deep inspiration is difficult, and when pain is present it naturally aggravates.

Expiration may be attended with the grunting-like sound as mentioned, or the respiratory act may be quiet or panting.

In many instances the increased rate of respiration is not attended with a great amount of oppression, while in others actual dyspnœa is marked. When pain is excessive it is an active factor. But dyspnœa occurs independently of pain, the cause being some irritation of the central nervous system from the toxæmia. It may also be due to the presence of extensive bronchial catarrh and to pulmonary œdema. In cases of extensive double pneumonia it may arise from actual loss of breathing surface.

Pain.—Pain in the side is a prominent, but not always constant, symptom. It may appear with the chill or just after or later. In many instances it is intense and each respiration is attended with a knife-like thrust. Coughing naturally adds to its intensity. There may be several areas of pain, the most common is below the nipple on the affected side. Sometimes it is beneath the scapula and sometimes it is referred to the abdomen. On the other hand, pain may be moderate or entirely absent. This is more likely to occur in asthenic cases.

Cough.—At first the cough is dry, unless there has been some preceding bronchitis. After the first twelve hours there is expectoration; the cough now becomes very frequent, persistent and harassing. It naturally aggravates the pain in the side. In aged persons, young children and alcoholics it may be absent. When the crisis appears it becomes easier and the expectoration freer.

The persistence of cough after convalescence is suggestive of some chronic change in the lung, and when it is of a paroxysmal nature and of great intensity it points to the possible presence of a pleural exudation.

The Sputum.—At first the sputum is mucoid and glairy, but after twenty-four hours it becomes tinged with blood. A marked feature is its tenacity. It will cling to the bottom of the sputum cup when it is inverted. The bright red coloring observed at first changes to a pinkish hue or a delicate rusty color. In some cases, especially asthenic, it may be brownish, the so-called prune-juice expectoration. The amount is variable, being from one or two to ten ounces in twenty-four hours. In children and old persons it may be absent, that is, none is expectorated. The presence, however, of secretion in the bronchial tubes is recognized and there may be cough, but no sputum is raised. In other instances it may be decidedly hæmorrhagic. This is an unfavorable sign. Some cases are ushered in with a decided hæmorrhage which must also be considered unfavorable.

Microscopic examination shows the sputum to consist of leucocytes, red blood-corpuscles in all stages of degeneration, mucous corpuscles and bronchial and alveolar epithelial cells. Hæmatoidin crystals are sometimes found. Casts of the alveoli and finer bronchi may be observed, the latter visible sometimes to the naked eye. Their presence in the rusty or pinkish sputum is pathognomonic. The rusty sputum alone is not so, as it may occur in acute miliary tuberculosis.

The Heart.—The heart-sounds are usually intensified. During the height of the fever bruits are common, especially in children. The possibility of endocarditis should be remembered in examining the heart.

The second pulmonic sound is accentuated. This is an important sign. As long as it remains it shows that the right side of the heart is able to empty itself freely. The enfeeblement of the second sound, on the other hand, indicates failing power of the right ventricle. It is accompanied by engorgement of the right side of the heart and increase of the area of dulness to the right of the sternum, the sounds being those of dilatation. In some cases, especially among aged and debilitated persons, there may be sudden failure of the heart early in the disease. This, however, is not necessarily fatal, as the patient may be able to respond to the administration of cardiac stimulants.

Nervous Symptoms.—Headache is common. It may be severe, congestive and greatly intensified by cough. On account of the pain the patient may dread every attack of coughing and will sometimes press the temples with both hands. Insomnia is a frequent symptom, but not more so than in other febrile affections. In many instances it is caused by the coughing rather than by nervous disturbances.

Ordinarily the mind remains clear, but delirium may occur. Its presence is significant of meningeal or nephritic complications. It is quite common in alcoholics, where it may be maniacal followed by coma. In such cases the true condition may be masked. Delirium may also appear during defervescence.

There is a form of mental aberration which in rare instances occurs during convalescence. This condition, as observed by the writer, exists for a few days when there is a return to the normal. Other observers mention a more prolonged disturbance.

The Urine.—The urine is generally reddish, of high specific gravity, highly acid and diminished in amount. There is decrease or absence of the chlorides. Their reappearance is a favorable sign, therefore tests are important aids to prognosis. They may be made as follows: Add to the specimen of urine a few drops of nitric acid, next add a similar quantity of a solution of nitrate of silver, a white clump-like precipitate forms. The precipitate thus produced will be dense and curdy, if the normal amount of chlorides are present, milky if lessened, and faint if almost or entirely absent. The urea and uric acid are usually augmented at first, but may decrease before the crisis, to increase again after its appearance. There is usually a small amount of albumin which disappears on recovery. Bile may sometimes be present, also casts denoting a commencing nephritis.

Gastro-Intestinal Symptoms.—Vomiting is not uncommon in the early stages, especially in children. There may also be a marked icteric hue. In some cases this may be the first symptom. The jaundice is generally of the catarrhal variety and the cases where it is present are usually serious. The tongue is dry and coated and, if a typhoid condition develops, is like that of typhoid fever. There is no desire for food. The bowels are generally constipated, but diarrhœa may sometimes be present. Meteorism may occasionally be a distressing and grave symptom.

The Skin.—Herpes is very common. Apparently it is more prevalent at some seasons than in others. Its usual seat is on the lips, but it may also appear on the nose, genitals or anus. It is said to be a favorable sign, but its real significance and its relation to the disease is not known. Perspirations during the course of the disease are common and when excessive tend to weaken the patient. They are indicative of the toxic process. At the crisis they may be very profuse. The cheeks are often flushed. Redness of one cheek to which allusion has been made is commonly observed on the same side with the pneumonic lung.

Epistaxis.—Epistaxis sometimes occurs without relation to the height of the fever and other symptoms. Except in the case of aged and feeble persons, and when excessive, it does not appear to have any serious significance.

The Crisis.—The crisis is an essential feature of pneumonia. It may make its appearance anywhere from the fifth to the twelfth day. It is said to be more frequent on odd days, but that is doubtful. The usual critical day is the seventh, eighth or ninth. In rare instances it is said to have occurred on the third and even the second day. Its appearance marks the end of the fever and amelioration of symptoms. The falling of the temperature at the crisis may occupy from six to forty-eight hours, that is, it may be by crisis (Figs. 42 and 43) or it may be by lysis (Fig. 45). Occasionally it is very abrupt, with symptoms of collapse, profuse perspiration and urination, vomiting, diarrhœa and hæmorrhage from the mucous membranes. In a fair proportion of cases the fall is by lysis covering a period of several days. In cases when the temperature persists until the twelfth day, this is usually the case.

PHYSICAL SIGNS.—*Inspection*, as far as thoracic symptoms are concerned, does not reveal important phenomena. The patient reclines on the back or affected side, well supported by pillows. Orthopnœa is not common. The other symptoms which are observable on inspection are mostly those which have been mentioned.

In the earliest period of the disease there is no visible change in the movements of the chest walls. Later diminished expansion is observed on the affected side, while there is a noticeable compensatory increase on the healthy side. The accessory muscles of respiration may be called into action and the nostrils may dilate with each inspiration. When that portion of the upper left lobe of the lung which overlaps the heart is involved, there may be increase in the area of visible cardiac impulse.

Palpation confirms the change of the movements of the chest walls. When hepatization has taken place, there is increase of vocal fremitus over the affected area. But when the bronchi are filled with thick mucus, or in massive pneumonia when they are occluded, there is loss of fremitus. Before attempting fremitus, the patient should be asked to cough.

Percussion shows valuable signs. In the first stage the note may be hollow or slightly tympanitic, the so-called Skodiac resonance. This quality is due to the relaxed condition of the lung which results from the engorgement, the walls of the alveoli losing their normal elasticity, being no longer firmly stretched as in health.

In the second stage there is dulness. The quality of the percussion-note, however, varies from a decidedly flat sound to one suggestive of tympany. The dulness never has the wooden quality observable in effusion. There is also a sense of resistance experienced by the pleximeter finger. The tympanitic quality in this stage is due to the fact that consolidation is incomplete and that the condition of relaxation of lung tissue as above mentioned exists. In cases where the consolidation is complete the vibration of air in the bronchi is said to be the cause of the tympany. Tympanitic resonance is also heard over the healthy lung adjacent to and over the consolidated area near the trachea and main bronchi. A cracked-pot note may be observed when areas of consolidation are in close proximity to relaxed lung tissue. An occasional change is Wintrich's sign which may be marked in pneumonia of the upper lobe. The healthy lung gives a higher resonant note. During resolution the normal percussion-note returns.

In the beginning of the last stage there is the same condition of percussion-note as observed in the second, but changes at once begin and the normal sound in favorable cases returns.

Auscultation reveals the most essential features of the physical signs of pneumonia. In the earliest periods of the first stage the respiratory sounds are harsh and feeble, but the patient is seldom seen at this time. As the engorgement becomes greater the crepitant râle is produced. This is the characteristic sign of inflammatory engorgement of the lungs. It denotes the first period of inflammatory action. The crepitant râle heard at the end of inspiration gives the impression of the bursting of very small air-bubbles of uniform size. It is probably due either to the separation of the agglutinated walls of the alveoli or to the effort of the bronchioles to empty into the alveoli through a tenacious fluid exudate. The latter is the most acceptable explanation. In association with these signs there may be those of pleurisy of varying degrees of intensity.

As the air-cells fill with the croupous exudate the crepitant râle disappears, the second stage becomes established and bronchial breathing takes its place. Bronchial breathing is characteristic of the second stage of pneumonia. Bronchophony is also audible. In pneumonia of the upper lobes and anterior surface of the lung it may be absent. This is explained as due to incomplete hepatization. A high-pitched and well-marked bronchophony is characteristic of pneumonia.

When there is pleural effusion the signs are necessarily obscured. Bronchophony is diminished or absent below the level of the effusion where ægophony may take its place.

The heart-sounds are transmitted over a wider area as the result of the consolidation. An important feature is accentuation of the pulmonic second sound, a natural result of the obstruction arising in the pulmonary circuit from the pulmonary engorgement. A phenomenon of interest is the occasional presence of amphonic respiration. This may be observed in the upper portion of the lung immediately below the clavicle and is usually associated with a metallic quality of the percussion-note.

As the third stage appears the signs on auscultation observed in the previous stages disappear in a retrogressive order. The bronchial breathing gives way to broncho-vesicular; subcrepitant râles appear, the râle redux, characteristic of the resolving stage of pneumonia. These gradually disappear and the normal vesicular breathing returns in about three weeks.

X-Ray Diagnosis, see Part VII.

RESOLUTION.—Convalescence is usually prompt and sequelæ infrequent. The products of inflammation disappear partly by absorption and partly by expectoration on coughing. The time occupied by the process of resolution varies. Sometimes the dulness and tubular breathing disappear in a week, sometimes it takes longer for the lung to completely clear. When there has been an extensive pleurisy a certain amount of dulness may persist from thickening of the pleura.

RELAPSES.—The recurrence of a pneumonic inflammation after the primary attack has passed the critical period is observed in rare instances. This phenomenon is more likely to occur between the tenth and sixteenth day.

DELAYED RESOLUTION.—After the crisis has passed and the temperature has been normal for a day or two there may occasionally be a return of the fever. This may arise simply from delayed or anomalous resolution, or it may be the precursor of some terminal complication. In all instances it is always a source of anxiety. While more frequent in the debilitated and infirm it is also observed in those who have previously been strong. It is claimed to be more common in pneumonia of the apex. There are several types of delayed resolution which may be classified under three general heads, as follows: (1) That in which the temperature has fallen to normal by crisis, yet the lung clears up very slowly, sometimes three weeks or more elapsing before the evidence of consolidation and râles disappear. (2) That in which the temperature falls by lysis, yet lingers on in a modified degree, accompanied by sweats and quick pulse. This condition may continue for several weeks or even two or three months and the lung yet resolve. (3) That in which the signs of consolidation persist to a marked and intense degree and the fever, although having fallen by lysis, continues to a certain extent. The tendency of this type is to secondary lesions, such as fibroid degeneration of the lung. In all forms of delayed resolution, notwithstanding the favorable termination which may be looked for in many instances, the possibility of secondary changes must never be overlooked.

TERMINATION IN NON-RESOLUTION.—The pathological conditions which may occur in the instances of non-resolution in pneumonia are abscess or purulent infiltration, gangrene, and, as mentioned, fibroid induration. Pulmonary tuberculosis does not immediately follow simple croupous pneumonia. Those cases in which it has apparently resulted have been tuberculous from the beginning.

PURULENT INFILTRATION OR ABSCESS.—When this condition follows the course of pneumonia there are sweats, sometimes rigors, protracted fever, either intermittent or remittent in character, cough, muco-purulent expectoration and general symptoms of hectic. There may be only one abscess or there may be a number. Grisolle, in twenty-nine cases collected from different writers, found that the abscesses are generally near the surface of the lung and except in rare instances are without a membrane.

GANGRENE.—This grave sequela is manifested by the horribly offensive odor which emanates from the patient's breath and sputum. It is attended with the symptoms of sepsis of varying degrees of intensity. Frequently the temperature curve does not run high. It is observed mostly in debilitated and broken-down constitutions and may occur with or without abscess-formation. It is generally associated with the saprophytic bacteria. The diagnosis can be made only when the characteristic symptoms appear after those of pneumonia.

CLINICAL VARIETIES.

Variation from the typical pneumonia may appear as follows:

Apex Pneumonia.—This form is said to be frequently characterized by the prominence of cerebral and asthenic symptoms. The cough and expectoration may be less marked than in other cases.

Central Pneumonia.—In this form the inflammatory process commences in the central portion of the lungs, so that the healthy lung tissue which intervenes obscures the true condition. The physiological signs may thus be absent for several days, but gradually they appear. Under these circumstances errors in the early diagnosis of the disease are common.

Double Pneumonia.—Pneumonia of both lungs presents no special features other than a more grave prognosis.

Massive Pneumonia.—This is a rare form in which the exudation fills the bronchi as well as the air-cells. The usual auscultatory signs are absent and the lung is flat as in pleural effusion. Moulds of the bronchi may be expectorated on coughing.

Migratory or Creeping Pneumonia.—In this variety, as the term implies, the disease is progressive, one portion of the lung being involved after another.

Pneumonia in Children.—The seat of lesion is more frequent in the apex of the lung in children than in adults. The first manifestations, as in adults, are sudden. In many instances they bear a strong resemblance to those of scarlet fever. There is high fever, pain in the affected side or abdomen, short cough, vomiting and diarrhœa. In children under three years of age convulsions are common; over that age they are rare. The convulsions may prove fatal before the pneumonia has fully developed. Convulsions may also occur during the course of the disease. They may be preceded by a period of excitement and followed by coma. There may also be rigidity and retraction of the neck. The resemblance to meningitis is marked and unless care is exercised error in diagnosis is likely to be made. Delirium is not uncommon and usually is present early, especially if the fever is high. Sputum is absent. Abdominal pain and retraction of the abdomen are frequent symptoms. The pupils may be contracted or dilated. The patient may toss and roll the head about; there may be involuntary action of the bladder and bowels; vomiting is common, the pulse rapid and respiration greatly accelerated, sometimes as high as eighty, and there may be Cheyne-Stokes respiration.

Pneumonia in the Aged.—In those advanced in years the onset of the disease is frequently insidious. The chill is often absent and the physical signs slow in developing and ill-defined. The temperature may not run high, frequently not mounting over 102° F. Expectoration is often absent. The constitutional symptoms are characterized by great prostration. Respiration, although frequently panting, is not so rapid as in younger persons, the pulse-rate is also lower, often below 100, frequently below 90. The temperature usually falls by lysis. Coma may occur; it may succeed delirium and precede death.

Pneumonia in Alcoholic Patients.—The onset may be either insidious or it may be attended with symptoms of decided virulence. In the first instance the symptoms are masked, being mostly those of a moderate degree of fever. In the latter there is high fever, cyanosis and early dyspnœa. The cases observed in the Metropolitan Hospital have usually been of this type. The not infrequent association of pneumonia with alcoholism in chronic drinkers should also be remembered when treating such cases.

Typhoid or Asthenic Pneumonia.—In this variety the symptoms, as the term implies, resemble those of typhoid fever. The toxic element predominates, sometimes appearing in the early periods of the disease, but usually at the termination of the first week. The pulmonary lesion bears no relation to the extent of the toxæmia, as the former may be limited, while the latter may be excessive. There is a dry, brown tongue, delirium or stupor and cough with prune-juice or hæmorrhagic expectoration. Gastro-intestinal symptoms are present, especially diarrhœa and meteorism. The toxæmia is doubtless due to virulent infection, both from pneumococcus of Fränkel and the streptococcus pyogenes.

Bilious Pneumonia.—In this form the chill is often prolonged and there are decided symptoms of hepatic derangement, such as jaundice, nausea and vomiting. These symptoms naturally increase the patient's distress and lend a more serious aspect to the condition.

Epidemic and Endemic Forms.—Allusion has been made to these varieties, examples of which are abundant in medical literature. When an epidemic type prevails the cases are usually characterized by certain types of variations, that is, they may show tendencies to the predominance of the virulence of toxæmia, to typhoid or gastro-intestinal symptoms or to complications such as endocarditis.

There is no doubt that many of the variations in the course of pneumonia depend upon the degree of virulence of the specific micro-organism and upon the virulence and development of the streptococcus pyogenes, but as yet we must acknowledge that clinically the bacteriology of pneumonia is still in the stage of development.

Ether Pneumonia.—This is simply a variety of aspiration pneumonia.

Larval, Abortive or Ephemeral Pneumonia.—Pneumonia also occurs in mild and abortive forms. The symptoms consist of a slight chill, followed by fever, with local signs of a somewhat indefinite character, yet suggestive of pneumonia. There is cough and possibly herpes. The symptoms may disappear in two or three days, when the condition is called abortive pneumonia. Sometimes they disappear in twenty-four hours, when it is called ephemeral pneumonia. In absence of post-mortem demonstration, such cases must always be open to question. It is doubtful whether a true pneumonic consolidation does occur. Some authors speak of a crisis on the first or second day. It seems to the writer that the use of the term is misleading under these conditions, as the disease is abortive and never reaches its full development.

Secondary Pneumonia.—As a secondary affection or complication of other diseases pneumonia is a factor of frequent occurrence and of grave significance. It is especially liable to occur in association with the infections, particularly influenza, typhoid fever, diphtheria and measles.

The ætiology of secondary pneumonia is not clear. It is regarded by some as the result of infection of the lung by the pneumococci, the patient having been put in a condition for their development by the previous infectious disease. Others maintain that the specific cause of the latter, that is, the bacteria, have the power of causing acute lobar pneumonia. Pulmonary infection by the bacillus coli communis, either from the intestinal or genito-urinary tract, is regarded by some writers as simply placing the patient in a condition favorable for the development of the micrococcus lanceolatus of Fränkel. The pathological changes are the same in character as in the other forms, but the areas involved are more irregularly distributed and the congestion surrounding the same is not infrequently excessive. On section of the lung there is found a mingling of croupous and broncho-pneumonia, the latter being closely situated areas giving the appearance of a true lobar consolidation. The surface is also smooth, but not so dry.

Microscopical examination shows a greater abundance of the cellular elements in the exudate and less of the fibrinous, the former infiltrating the alveolar walls.

Bacteriological examination reveals the micrococcus lanceolatus, the streptococcus pyogenes, the staphylococcus and the bacillus coli communis.

It has been suggested that this form of pneumonia should be recognized as a definite type under the appellation of acute cellular pneumonia (Finkler).

The clinical manifestations of secondary pneumonia may lack distinctive features and the condition remains latent and undefined. There is usually, however, some elevation of temperature and increase in the rate of respiration. In some instances, on the other hand, there may be decided symptoms in the form of rigors, marked increase of temperature and respiration, pain, cough and the signs of pulmonic consolidation.

Terminal Pneumonia.—This form might well be considered secondary. It occurs usually in winter in persons with chronic tuberculosis, Bright's disease, diabetes and cardiac insufficiency, as a termination of these affections and direct cause of death. There may be only a slight elevation of temperature and increase of respiration. In many cases the condition remains unrecognized. Physical examination would reveal the signs of pulmonary consolidation, but the state of the patient being that of *in extremis*, this is often omitted and the condition of the lungs not discovered until autopsy. In some cases abscess or gangrene may develop, especially in diabetes.

Embolic Pneumonia.—This occurs in both the non-septic and septic forms. These conditions are discussed under the heads of Hæmorrhagic Infarct of the Lung and Pulmonary Metastatic Abscess.

COMPLICATIONS.

The most important complications are as follows:

Pleurisy.—When the pneumonic process is situated near the surface of the lung there is necessarily more or less pleuritic inflammation which, strictly speaking, is part of the affection rather than a complication. Aside from the pain to which it gives rise, this condition is not of serious significance, resolving when the pneumonia resolves. There are, however, cases in which the pleurisy predominates and when the term pleuro-pneumonia best expresses the true condition. In these instances the exudation may be serofibrinous with a copious effusion, but differs from the ordinary acute pleurisy in that the amount of fibrin is larger and sometimes found in thick, tough, curd-like layers. The presence of pleurisy on the side opposite to that on which the pneumonia is seated is an anomalous condition sometimes encountered. The difficulties of diagnosis are apparent.

Bacteriological examination of the effusion shows the presence of the pneumococci. The pleuritic inflammation may result in empyema. If the process is caused by the streptococci the condition usually terminates fatally. The effusion may not be recognized during the height of the pneumonic fever, but after the disease has run its course and the fever has subsided, if then a rise occurs followed by irregular exacerbations and the dulness persists, purulent effusion may be suspected. The respiration is feeble and there is absence of râles. These phenomena may be observed to a certain extent in pleural thickening, but the diagnosis may be made by exploration with the hypodermic needle. Later dyspnœa and cyanosis may develop.

In some instances pleurisy appears at the end of pneumonia as a terminal complication. Under these conditions it is more serious.

Pericarditis.—This is more common in pneumonia of the left lung and in double pneumonia. It is always a serious complication, especially with children, where it is more frequent than with adults and more often fatal. The process may be either plastic or serofibrinous, although usually the former. The condition is recognized by the friction murmur, although there may be difficulty in determining the same when that portion of the lung which overlies the pericardium is involved in the pneumonic inflammation.

Increased difficulty in breathing, small, rapid and sometimes irregular pulse and gradual suppression of heart-sounds will appear when pericardial effusion is marked. In some cases the effusion becomes purulent.

Notwithstanding the gravity which the presence of pericarditis lends to pneumonia, recovery frequently takes place even in the most serious cases.

Endocarditis.—The occurrence of endocarditis with pneumonia is much more common than was formerly supposed. The presence of the pneumococcus in the products of endocardial inflammation strongly suggest the probability that these organisms infect the endocardium as well as the lungs. Their exact relation, however, to endocarditis is as yet undetermined. The left side of the heart is generally involved in preference to the right.

The malignant form of the disease is rather more frequent than the benign. The presence of an old endocarditis is obviously a predisposing element. The intensity of the pneumonic inflammation is not necessarily a criterion of the liability of the complication of the endocardium.

The presence of pneumonic endocarditis is often not recognized, partly because of the latency and obscurity of its symptoms and partly because of the preponderance of those of pneumonia. In some cases the symptoms are pronounced. There is præcordial distress, sometimes slight, dull pain and the presence of murmurs of recent formation, and rigors and sweats. The fever is protracted, but the temperature curve may not be excessively high notwithstanding the evidences of sepsis. Sometimes embolic processes appear in the course of the disease, especially in the instance of malignant endocarditis after the crisis. Meningitis is not infrequently associated with these cases and is usually the cause of cerebral symptoms under these conditions.

Jaundice.—The association of jaundice and pneumonia has been the subject of discussion and variation of opinion. Some observers distinguish between an icteroid pneumonia and one with true jaundice. There does not seem to be, however, a well-established connection between the two affections, except that in persons subject to hepatic derangements and excesses in drinking bilious symptoms are liable to appear.

The presence of jaundice with pneumonia does not necessarily render the prognosis unfavorable. But when pneumonia arises secondarily to jaundice, that is, not dependent upon the diplococcus, but upon other bacteria, the outlook is much more unfavorable. Compare Bilious Pneumonia and Gastro-Intestinal Symptoms.

Myocarditis.—This is an unusual complication. It may appear with endocarditis.

Meningitis.—This is a complication of much gravity. It generally appears during the height of the fever and may be mistaken for delirium. In some cases it may develop later when it is more readily recognized. Its association with endocarditis has already been mentioned.

Arthritis.—The presence of joint inflammation is a complication sometimes encountered. It may appear before the pulmonic process or a few days after. In the recorded cases many were associated with peri- and endocarditis and pleurisy. Suppuration of the joints was common.

Parotitis.—This occasionally occurs, especially when there are endocardial complications.

Otitis Media.—May sometimes develop, especially in children.

PNEUMONIA ASSOCIATED WITH OTHER DISEASES.—By this is meant the presence of pneumonia with other affections, not of secondary origin. Malaria is mentioned by some writers and a type called malarial has been described, but the claim is not substantiated. Malaria, however, may be associated with pneumonia and greatly influence its course.

Emphysema favors the development of pneumonia and greatly adds to its danger. Chronic nephritis is also a very unfavorable factor. The presence of valvular disease, although unfavorable, is less so.

The association of pneumonia with tuberculosis may occasionally be observed. Apart from that form of pulmonary tuberculosis called galloping or pneumonic tuberculosis, an acute pneumonic inflammation may occur superimposed upon the chronic condition and usually end fatally. The condition is attended with high fever, dyspnœa, flushed face, sweats, and sometimes hæmorrhages.

Diagnosis.—When following a typical course there is probably no disease more readily recognized than croupous pneumonia. The sudden onset of chill, the fever, the cough, the blood-tinged or rusty sputum, the quickened respiration, the leucocytosis, the crepitant râle in the first stage and bronchial breathing in the second do not permit of error in diagnosis.

When pneumonia begins in the central portion of the lung the physical signs may be late in developing, and, unless care is exercised, valuable time may be lost before the real danger is appreciated. In pneumonia of the apex and upper lobe the characteristic symptoms and signs may likewise be slow in developing.

In children, in the aged, in alcoholics, and in its secondary manifestations, pneumonia is frequently difficult to recognize. In children the cerebral symptoms which so frequently accompany the pneumonic process may be of such a marked degree of intensity that the affection may closely simulate meningitis. Convulsions, coma and the absence of expectoration lend additional difficulties.

In these cases a careful examination of the lungs will constitute the sole means of differentiation.

Among elderly persons the subtleness of the onset and absence of definite symptoms may likewise prove a source of error. Here, again, the recognition of the condition will rest on the physical signs. In aged persons when the symptoms of bronchitis, fever and prostration are present, even when the physical signs are indefinite, the possibility of the development of pneumonia should always be borne in mind.

Secondary pneumonia can be recognized by the presence of rusty sputum and the physical signs. When these are absent the diagnosis is uncertain and difficult. Sudden rise of temperature and increased rapidity of respiration and pulse in the course of such affections as typhoid and other infections and in the advanced stages of diabetes, pulmonary tuberculosis and chronic nephritis, suggest the presence of pneumonia.

Pleurisy with effusion, acute tuberculosis, broncho-pneumonia, typhoid fever and pneumo-typhoid also lend difficulties to the diagnosis in some instances. Pleurisy with effusion is more likely to prove a source of error than any other affection, especially in children in whom in both pneumonia and pleurisy there may be bronchial breathing and increased tactile fremitus. But in pleurisy the percussion-note is usually more wooden in character and there may be ægophony. These signs, however, are not wholly reliable. In uncertain cases the diagnosis can only be made by the exploratory needle. The displacement of the neighboring organs, namely, the liver, heart and spleen, as observed in pleurisy with effusion, will assist in the diagnosis. At the same time it should not be forgotten that such displacements are often absent.

Pneumonia is scarcely likely to be confused with chronic pulmonary tuberculosis, but in regard to acute pulmonic tuberculosis it is another matter. A chill may mark the onset of both, but is not so constant a symptom as in pneumonia. The sputum of acute tuberculosis is more likely to be hæmorrhagic and the temperature curve more distinctly that of tuberculosis, but neither of these symptoms is conclusive. The only reliable means of diagnosis is a microscopic examination of the sputum to determine whether the tubercle bacillus or diplococcus pneumoniæ is present. If the period of the crisis of pneumonia should have passed, the condition of the patient at that time will prove an additional source of diagnosis.

When pneumonia is situated in the apex of the lung and many of its characteristic symptoms, such as rusty sputum, bronchial breathing and marked dulness, are absent, the diagnosis between it and acute tuberculosis is extremely difficult. Especially is this the case when the temperature curves of both are similar. The diagnosis in such cases will rest mainly upon the examination of the sputum. Although the tubercle bacilli and elastic tissue may not be found until late in the development of the acute tubercular process, the history will always furnish a valuable aid in these difficult cases.

Croupous pneumonia can always be readily distinguished from broncho-pneumonia when appearing in its typical form. The latter is more gradual in its development. The physical signs show bilateral involvement of the lungs; the areas of consolidation are smaller and the upper lobe is more generally involved than in croupous pneumonia. There are also the râles of bronchitis, both fine and coarse, and absence of well-marked tubular breathing, although this may develop later. There is likewise usually more dyspnœa and greater rapidity of respiration and greater cyanosis. The sputum is seldom blood streaked and the course of the disease is prolonged and not marked by crisis. Broncho-pneumonia is also much more frequently associated secondarily with some other disease, such as measles.

When a pneumonic patient falls into a typhoid state and is seen for the first time in that condition, the diagnosis is difficult. If it is possible to obtain the history of the case, a differentiation can usually be readily made, but sometimes this is impossible, especially in hospital practice. Under these conditions the question will rest mainly upon the Widal and diazo tests.

There are cases which with aptness have been called pneumotyphoid, that is, those where the pneumonic consolidation is apparently coincident with the earlier manifestations of typhoid symptoms. In these the diagnosis is also difficult and will likewise rest upon the Widal and diazo tests.

Pneumonia is sometimes mistaken for appendicitis. It is needless to say that a proper examination removes the difficulties.

In all cases of sudden chill the chest should be examined. Sudden chill with pain in the side always suggests pneumonia. Although it cannot be regarded as diagnostic, as the same symptoms may be observed in mastitis and in pleurodynia. In all instances it is important that all portions of the chest should be included in the examination, as râles are frequently first heard in the lateral and axillary areas in pneumonia of the lower lobes.

The differentiation between pneumonia and pulmonary œdema, pulmonary infarction, hypostatic congestion, is discussed under those subjects.

Prognosis.—Of all acute diseases pneumonia is the most frequent cause of death, and of all diseases, both acute and chronic, it is the second only to tuberculosis in the destruction of human life. The percentages of death in different statistical groups show marked variations, ranging all the way from 6 to 40 per cent. This wide divergence is explainable in part to the difference in severity of the prevailing types of the disease and in part to the methods of treatment, for there is no doubt that the large doses of quinine, morphine and venesection in vogue at one time gave results vastly inferior to those of treatment according to the methods of the new school of medicine. It is said that the disease is more fatal among negroes than among white races.

Under proper care the majority of patients recover, yet the outlook in every case of croupous pneumonia is always grave and uncertain. The influences which bear strongly upon the result are the virulence of the toxæmia, age, previous habits of life, general health, presence of complications and of the disease to which the pneumonic inflammation is secondary. Toxæmia is by far the most important element. In the majority of cases it is much more so than the amount of consolidation and the pyrexia. The prostration, rapidity of respiration and heart action are the chief indicators of the extent of the toxic elements. It should be recalled in this connection that the amount of lung tissue involved beàrs no relation to the toxic infection, which may be excessive when the inflamed area is small or mild when it is extensive. It must not, however, be inferred that the extent of the implicated area has no influence on the prognosis, for when it is excessive the danger to life is greatly increased.

Age shows its influence as follows: In early childhood and old age the prognosis is especially unfavorable. Under one year of age it is more fatal than two or three, the fatality decreasing as the age advances. After fifty the death-rate again advances, and at sixty it is very high. Generally speaking, the outlook may be said to be much more favorable in the young. Habits of life and the condition of the general health, as in other acute affections, must always be taken into consideration. Persons addicted to excesses of any kind, especially that of alcohol and high living, more easily fall victims. Likewise those who in addition to being heavy drinkers have lived poorly both as to food and hygiene. Examples of the latter are frequent in public hospitals among men in early middle life. In short, pneumonia is much more fatal among those who have used up or abused, so to speak, their reserve forces or power of resistance.

Complications necessarily add to the danger. With meningitis it is generally fatal; endocarditis, while very grave, is not necessarily fatal. Pericarditis is also serious, especially in children when it is often the cause of death.

As a secondary affection pneumonia in the greater proportion of cases terminates the case.

The following symptoms are unfavorable: A persistency of high temperature, 104° F. and over; high fever, especially in the morning, as, for example, 104° F.; an exacerbation of fever on the fourth day and absence of the pseudo-crisis; a pulse of over 120; disturbances of the pulse rhythm; rapid development of consolidation; large quantities of frothy expectoration; hæmorrhagic expectoration; a respiratory rate of 50 or more; a rise of temperature and increased frequency of the pulse on or after the seventh day; continued delirium; pneumococci in the blood and offensive sputum. Cases beginning with gastro-intestinal symptoms are much more unfavorable than those which begin with a chill. The amount of albumin appears to bear a direct relation to the prognosis.

In fatal cases the majority of deaths occur during the stage of red hepatization just before the crisis; a few occur before. Pneumonia at the apex is considered more serious than that of the lower lobes. This is denied by Grisolle. Pneumonia of the right lobe is generally thought more favorable than that of the left, because the liability of pericardial implication by extension is less.

be viewed impartially from all sides. In a disease which is self-limited and which may terminate naturally by crisis as early as the third or fourth day, and again which differs in its virulence in different seasons, it is manifestly a difficult question to determine the merits of any one line of treatment.

The following are the most important remedies.

Veratrum viride is of primary importance in the earliest stages when there is high temperature, strong, full pulse, marked pulmonary engorgement and the patient is inclined to be quiet and calm.

The dose should fall short of the depressant action of the drug. Hale recommends two drops of the tincture every half-hour in urgent cases. The writer recommends a quarter to half a drop in ordinary cases half-hourly or hourly.

Aconitum is also called for in the early stages. It should be preferred to veratrum viride when in addition to the presence of high fever and pulmonary engorgement there is great restlessness and anxiety, sense of weight about the chest, hard, full pulse, headache and flushed face. It is best suited to sthenic cases when a high fever follows a sharp, well-defined chill. It should be administered in small doses of the tincture, a third or a quarter of a minim hourly or half-hourly.

Bryonia is the most important drug in the second stage when the restlessness has passed. There may, however, be anxiety due to the dyspnœa. The presence of sharp, cutting pleuritic pains and pleural complications are special indications. Scanty expectoration, white-coated tongue, dryness of the mouth, thirst, disordered stomach, engorgement of the liver, constipation, scanty urine, hard, tense pulse are important indications.

Bryonia and veratrum viride or aconite may be given advantageously in alternation half-hourly or hourly.

Ferrum phosphoricum is a favorite remedy among many practitioners for asthenic cases when the patient's constitution is enfeebled from any cause. Hence in the aged, debilitated and cachectic it finds its sphere. It is also of service in pneumonia arising secondarily in association with infectious diseases. The symptoms which especially indicate its administration are mild chill and less pronounced and rapid rise of temperature, full, soft pulse, rusty or bloody expectoration, early absence of nervous excitement, listlessness, apathy and drowsiness. Bronchial complications are an additional indication, while marked pleurisy is a contraindication. The remedy should not be used in sthenic cases.

Antimonium tartaratum, which has been condemned by the old school, is most useful when administered in proper doses in those cases characterized by asthenia, both in the old and young, bronchial complications with rattling cough and loud râles in the large tubes and numerous subcrepitant râles, the presence of much mucus, but little expectoration, copious cold perspiration, cyanosis, prostration, great anxiety, restlessness and tendency to pulmonary œdema. Cases complicated with decided bronchitis, with profuse mucous secretion and inability to expectorate and consequent embarrassment of the lungs from the excessive accumulation of mucus, are those in which this remedy is especially indicated. Asthenic cases of pneumonia, those secondary to influenza, measles, etc., are usually also benefited by tartar emetic. The dose should be two grains of the second decimal, that is, one-fiftieth of a grain, every two hours.

Iodinum is recommended in the stage of consolidation in place of bryonia in so-called scrofulous patients with soft flesh and tendency to glandular enlargement. High temperature, absence of pleuritic pains are said to point to its use, which appears to be somewhat limited in spite of warm endorsement of its good effects. The remedy is best used in one- or two-drop doses of the second decimal dilution.

Phosphorus is an important remedy of the second stage. It owes its prominence to Fleischman of Vienna. By general consent it is considered as indicated in cases which deviate from the regular course of the disease. It is best suited to delicate persons, those advanced in years and those in whom there is much exhaustion and feebleness. Its special indications are prostration, great oppression of the chest, dyspnœa, bloody muco-purulent sputum, pain not intense, or vague stitches, consolidation marked, especially of lower half of right lung, mucous râles extensive, fever not marked and expectoration difficult. Tendency to run into the typhoid state is an additional and important indication for its use. It also should be considered in so-called bilious pneumonia. Care should be exercised to obtain a reliable preparation, as many are worthless.

Antimonium arseniatum is useful in pneumonia following influenza in elderly patients with severe dyspnœa, loud mucous rattling, frothy mucus, with difficult expectoration, feeble pulse and symptoms of failing vitality.

Sulphur is important when resolution is delayed. When the second stage is apparently prolonged, that is, the eighth day has passed, and the symptoms remain active and there is great vascular excitement, sulphur is said to hasten to the crisis. Again, when the crisis is passed, but the patient's condition remains unchanged, the lung solid and convalescence retarded, sulphur may be administered with good results. The cases in which this remedy appears to act best are those where the consolidation is marked and rapid. Also in pneumonia of the aged and in that of children when the condition simulates cerebral meningitis or is actually accompanied by it, with symptoms of rolling of the head, strabismus, coma, etc. Forenoon aggravation, sensation of heat in the chest, flushing of the face and irritability are further indications.

Chelidonium major is of service in bilious pneumonia and that of children when the lower right lobe is involved. Special indications are pain under the inferior angle of the right scapula, bilious symptoms, such as tendency to jaundice, vomiting and diarrhœa with high temperature.

Mercurius is likewise of use in bilious pneumonia with bronchial symptoms and when the process is on the right side. The remedy appears indicated when the fever has subsided, but dyspnœa and pain with bilious symptoms continue.

Hepar sulphuris is limited to the condition of non-resolution marked by a tendency to purulent infiltration and abscess. Symptoms indicating purulency, that is, fever, emaciation, purulent, fetid sputum, are its chief indications. Extreme sensitiveness to the air, profuse secretion and easily excited perspiration are also indications.

Of the above remedies veratum viride and bryonia are without doubt the most important and generally applicable in typical cases. The former controls the fever when properly administered, without depressant action. Of this there is no question. The latter exerts an ameliorating influence which, if the remedy is acting favorably, should manifest itself on the third day. It is the writer's custom to give veratrum viride and bryonia in alternation in the first stage. If there is no amelioration under bryonia on the third or fourth day the remedy should be replaced and another given according to indications. If, on the other hand, the dyspnœa, fever and pulse-rate continue to decrease steadily, the remedy should be continued until the crisis.

Oxygen is of service when there are evidences of cardiac and respiratory failure with cyanosis, pulmonary œdema, extensive consolidation and bronchitis. It should be administered freely according to indications and will generally, at least temporarily, relieve dyspnœa.

Creasotum has been employed to some extent in the treatment of pneumonia. Theoretically, in view of the infectious nature of the disease, it should do good service and the very favorable reports seem to confirm this opinion. The writer has employed it with beneficial results in a number of cases, but more extended use is necessary before any definite conclusion can be reached. It is best given in the form of the carbonate, fifteen grains every three hours. The following is a suitable formula :

℞.	Creasoti carbonatis,	℥ss.
	Glycerini,	℥j.
	Syr. simplicis,	℥viij.

M. Sig.—A tablespoonful every three hours.

The drug is usually well tolerated. It produces, however, an unpleasant taste and sensation of dryness in the mouth. It should be given from the beginning of the disease to the crisis.

Digitalis, according to the claim of Petrescu,[*] of Bucharest, is considered especially efficacious in pneumonia. Enormous doses were given and very satisfactory results claimed. Up to 1891 Petrescu had eight hundred and twenty-five cases with a mortality of 2.06 per cent. On the other hand, Risell had one hundred and twenty-seven cases treated without digitalis with a mortality of 1.8 per cent. While the observations of Petrescu met for a time with a favorable endorsement, subsequently other observers[†] brought reports of an unfavorable nature. Von Jürgensen does not regard digitalis as a specific in pneumonia, but in cases where the patient has previously suffered from cardiac weakness or when in the course of the disease cardiac insufficiency develops, and the pulse becomes greatly accelerated and at the same time irregular, digitalis is indicated. The remedy is mentioned because of the favorable results once claimed for it.

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[*] Revue de medecine, 1893, xiii., 196. Sur la Traitement de la Pneumonie par la Digitale à haute doses ou dose therapeutiques, par M. le docteur Z. Petrescu.

[†] Nothnagel's Encyclopedia of Practical Medicine, Diseases of the Lungs, Bronchi and Pleuræ, Philadelphia, 1903.

Serum Therapy.—Interesting and remarkable results have been obtained by the brothers Klemperer, Fowlinsky, Emmerich, Netter and others with anti-pneumococci serum. Netter succeeded in immunizing rabbits against the pneumococci, employing the dried spleen of animals infected with the same, old pleuritic exudate containing pneumococci and the sputum of convalescents after the crisis, when it is no longer virulent. F. and G. Klemperer injected into two rabbits 20 c.c. of a purulent exudate from the pleura which had been freed from other germs by repeated cultivation. Both were inoculated fourteen days after and remained free from infection. The control animal succumbed.

The conclusion of their experiments demonstrates that any culture medium in which the pneumococcus has grown will render immune an animal against pneumonic septicæmia even after the cocci have been removed; that the culture serum does not kill the cocci, but renders harmless the virulence of the products formed by the pneumococci.

The Klemperer brothers injected themselves with 0.5 to 3 c.c. of serum taken from animals rendered immune against pneumonia infection and demonstrated the innocuousness of the substances. They therefore injected from 4 to 6 c.c. of serum in six pneumonia patients. In all the cases in from six to twelve hours after injection a decided fall of temperature occurred, with slowing of the pulse and respiration.

Other experimenters have reported results on this same line. It is impossible, however, to draw definite conclusions, as the application of serum therapy to pneumonia has as yet been too limited.

TREATMENT OF SPECIAL CONDITIONS.

Cough.—There is little that can be done for the cough of pneumonia, for it is important that expectoration should be encouraged.

A mixture of lemon-juice and glycerin, half an ounce each with sufficient water to make two ounces, may prove a useful palliative in one-drachm doses.

Pain.—For the relief of the pleuritic pain which is frequently so severe in the early stages, in addition to the internal administration of bryonia, a small poultice of flaxseed meal may be applied under the pneumonia jacket and renewed at frequent intervals. Warmed laudanum may be poured over the poultice. In some instances the ice-bag may be used with better results, especially in cases of a sthenic type. Hot wet compresses should be avoided, as they are apt to wet the clothing and the bed.

Nervous Excitement and Delirium.—When the patient shows a high stage of nervous excitement, if it be due to fever, employ cold sponging; if from sleeplessness, pain, severe headache, a small dose of morphine may be administered. If there is decided delirium, hyoscyamus, belladonna, agaricin and hyoscine hydrobromate, as mentioned under the recommendations for typhoid pneumonia, should be considered.

Hyperpyrexia.—As the persistent presence of high fever tends to disorganize the blood and to paralyze the nerve centres, this condition calls for interference as a dangerous symptom.

In many instances veratrum viride, aconite and bryonia will control the fever to a reasonable and safe degree. When these fail recourse should be had to sponging, sponging limb by limb with water at 70° to 75° F. as mentioned. Another method is the application of broad, flat ice-bags to the affected side, or in their absence ice wrapped in oiled silk.

In extreme cases of hyperpyrexia with embarrassment of the circulation, cold packs have been used with beneficial results, sheets wrung out in water at 65° F. being applied.

Baruch recommends for children a tub-bath at 95° F. with friction, the water being gradually cooled to 85° F.

The coal-tar derivatives are generally condemned, but phenalgin and phenacetin, if given in two-and-a-half to three-grain doses, will often lower the temperature without untoward effects.

The Onset of Heart Weakness.—Failure of the heart is principally due to the action of toxins. An additional cause is continuously high fever. While it is obviously impossible to separate these influences, it is important to combat the depressive effect of the toxæmia by the use of stimulants according to indications.

When the Heart is Failing.—When the pulse becomes weak, irregular and intermittent, the respiration shorter, and cyanosis appears with evidences of pulmonary œdema in the uninvolved portion of the lung, prompt action is necessary in order, if possible, to tide the patient over this dangerous point. The amount of alcoholic stimulants should be at once increased. Frequently champagne will prove most effective. It should be given freely. In this emergency inhalation of oxygen gas should be administered.

Of medicinal cardiac stimulants strychnia is most extensively employed. It is best administered hypodermically in $\frac{1}{100}$ to $\frac{1}{20}$-grain doses every three to six hours according to indications. Goodno recommended agaracin 1x in one- to three-grain doses every two or three hours. Nitro-glycerin in one-drop doses of the 1-per-cent. solution hypodermically every half to every three hours is an important agent, especially when engorgement of the right side of the heart is a prominent symptom.

It may be given every half hour for a few doses, but when its use is continued, it may be given every three hours. It should be remembered that the effects of the remedy are not lasting.

Digitalis is not so frequently employed for the failing heart of pneumonia, but it will be found effective when the pulse suddenly becomes quick, irregular and intermittent. It may be given in ten-drop doses of the tincture every two hours for several doses.

Other cardiac stimulants are caffein, ether, sterilized camphorated oil hypodermically, and aromatic spirits of ammonia and musk by mouth.

When collateral pulmonary œdema is threatened in sthenic patients and there is marked embarrassment of the pulmonary circulation and frothy expectoration, venesection is advocated by some observers. Dry cupping, mustard poultices should also be considered in this condition.

The Typhoid Condition.—When pneumonia tends to assume typhoid symptoms another line of medication must be adopted. The principal remedies are rhus, hyoscyamus, arsenicum and baptisia.

Delirium.—Cerebral symptoms characterized by mild or active delirium call for the consideration of the following :

Hyoscyamus is of service in both mild and violent forms of delirium. The cerebral symptoms amenable to hyoscyamus are those which arise from the toxins and not from inflammatory action. Special indications: hallucinations, will not remain in bed, picking the bed clothes, fears of poisoning, constant muttering, mania with ridiculous gestures, complete loss of consciousness, when spoken to answers, but stupor and delirium return.

Belladonna, for active delirium with cerebral engorgement or inflammation, and in children for the convulsions. Special indications are dilated pupils, congestion of conjunctiva, strong pulsation of carotids, full, hard, frequent pulse, head drawn back, intolerance of noise, sensitiveness to light, anxiety, fear, dry cough, and loquaciousness.

Agaricus, for delirium of marked intensity, especially when accompanied by heart-weakness. In the pneumonia of alcoholics with violent delirium, constant raving and great exertion of power, it is a prominent drug. The conditions calling for its use, like hyoscyamus, are those which arise from toxæmia and not inflammation. The tincture should be used in two-drop doses or the alkaloid in one-grain doses of the first decimal trituration.

Hyoscine hydrobromate may be used in these cases of violent delirium when other means fail.

ASPIRATION PNEUMONIA.

SYNONYM.—*Deglutition Pneumonia.*

Aspiration pneumonia is that form of pneumonic process which results from the accidental entrance of fluid or other foreign matter into the bronchial tubes. It is considered by some writers as a form of catarrhal pneumonia, but differs pathologically and clinically.

Ætiology.—The disease arises in conditions of paralysis of the throat and respiratory tract from any cause which permits the aspiration or drawing into the lungs of any foreign matter, either in the form of oral or tracheal secretions or of small particles of food. It may occur in coma from apoplexy, ether anæsthesia and partial suffocation from drowning, as when persons have been under water sufficiently long to become unconscious and swallow more or less water which, together with the secretions of the mouth, passes into the lungs. Local affections of the bronchi with ulceration and perforation of the trachea, such as carcinomata, is another cause.

Cutting of the vagi will produce the same effect. Cases of carbolic acid poisoning in which some of the acid enters the respiratory tract constitute still another cause. Other irritating substances might obviously produce the same effect.

A component and essential factor in the causation of the disease is the presence of micro-organisms which accompany the foreign matter as it enters the respiratory tract. The infection occurring without or from the oral secretions.

The question as to whether the administration of ether as an anæsthetic results in pneumonia is one which has been the subject of some discussion. The observations of different anæsthetists differ. In Germany the administration of ether has been restricted by some clinicians on account of its injurious effects upon the respiratory tract. It has been suggested that the lowering of the vitality and chilling of the surfaces of the bronchi and alveoli incident to prolonged etherization places the parts in a condition more susceptible to infection from the inhaled air, disinfection of which has been suggested, or to infection from the inhalation of material containing micro-organisms. The use of nitrous oxide gas preliminary to etherization and the mixture of oxygen tend to lessen the liability to ether pneumonia.

Retention of bronchial secretion in semi-paralyzed states may act in the same way. Rupture of an empyema into the lung, aspiration of material from a bronchiectatic or tubercular cavity and hæmoptysis may likewise act as causes. Aspiration pneumonia also occurs in the newborn when the mother is septic. In these instances the infection of the infant can only be explained by the great virulence of the micro-organism.

Morbid Anatomy.—The distinctive character of aspiration pneumonia has been a matter of some dispute. E. Aufrecht* gives the following description: The changes are seated principally in the alveoli and the inter-alveolar tissue, while the finer bronchi, especially their vessels, appear to be unaffected. In catarrhal pneumonia, on the other hand, the process involves especially the finer bronchi and spreads to the alveoli. Aspiration pneumonia is further characterized by necrotic areas which, according to the nature of the bacteria present, lead either to gangrene or to abscess. The macroscopic examination of the lungs after death shows the presence of small lobular areas more in the dependent portions, which at the first glance appear like the process of catarrhal pneumonia, but on a closer examination disclose a difference in that they are more of a pale gray color, less firm in consistence, while their peripheral sections contain some air, the centres being consolidated. The tissues surrounding the gray areas are markedly congested. The process is not necessarily confined to the lower portion of the lung, but may be found more or less widely distributed and in both lungs.

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* Nothnagel's Encyclopedia of Medicine, Philadelphia, 1903.

The microscopical appearances, to quote Aufrecht again, are as follows: In quite a number of the bronchi there are amorphous masses—probably the result of disintegration of the leucocytes, the essential features of the disease. The alveoli are always the seat of the affection and frequently the inter-alveolar septa as well. If the disease takes a rapid course swelling of the alveolar epithelia with disintegration of their nuclei occurs, accompanied by intense hyperæmia of the alveolar capillaries, or if the course is not rapid, there is subsequently an exudation of white blood-corpuscles into the alveoli most affected, completely filling the same. Under such conditions a peripheral inflamed zone develops, the alveoli of which contain distended epithelia and many red and a few white blood-corpuscles. Between the cellular formation only a small amount of fibrin is found. When the intensity and duration of the process permit of its full development, the central alveoli which are congested with white blood-corpuscles are separated and cast off. This change may take place rapidly.* In such instances it is not difficult to comprehend that abscess formation may occur.

Changes in the pleura may likewise develop in the form of fibrinous exudation with hæmorrhage corresponding to the pulmonary foci in the interior of the lung. The exudations may be also serous and later become purulent. Again, a purulent focus may rupture into the pleural cavity.

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* Nothnagel's Encyclopedia of Medicine, Philadelphia, 1903.

Symptoms.—The symptoms of aspiration pneumonia are somewhat difficult to describe, owing partly to the fact that the disease is not recognized by all writers as a distinct process and in part to the nature of the conditions which frequently attend its development. Moreover, many of the factors in its ætiology imply a pre-existing morbid state. This of necessity influences or masks the manifestations of symptoms; as, for example, coma from apoplexy with more or less paralysis of the larynx and pharynx, or cancerous ulceration of the trachea. On the other hand, with submersion in any fluid matter aspiration of the same generally occurs in a previously healthy person.

Cough, expectoration, dyspnœa and fever are the essential symptoms, their development depending upon the duration of the disease.

Cough is not generally prominent unless the disease goes on to necrotic changes in the lung when it becomes severe. At first it is dry, but later as the lung breaks down it is attended with a purulent and offensive sputum. Dyspnœa is as a rule not excessive or urgent, fever may be high or moderate, in some and even fatal cases it has been absent.

Severe pleuritic pain occurs in instances. Endocardial changes may take place. In a case of the writer's from asphyxiation by illuminating gas, the patient died of gangrene following embolism of the anterior tibial artery. The course of the disease is usually very rapid whether it terminates in recovery or death.

PHYSICAL SIGNS.—The most important and significant evidences of aspiration pneumonia are those obtained by physical examination of the chest. There is dulness on percussion, and on auscultation crepitant râles and bronchial respiration is observed. These changes appear in areas which vary, but are commonly found in the lower and middle lobes.

Diagnosis.—In order to recognize aspiration pneumonia and separate it clinically from the catarrhal variety, it is necessary to take the history into consideration. This is due not alone to the indefinite character of the symptoms, but also to the fact that the objective symptoms of the two conditions do not at the onset materially differ.

Catarrhal pneumonia, excepting that secondary to influenza, is mostly a disease of childhood, while aspiration pneumonia is more common in adult life. Again, catarrhal pneumonia is generally secondary to some well recognized acute disease or chronic morbid state and in rare instances is primary. Aspiration pneumonia, on the other hand, is secondary to an entirely different class of morbid condition, as mentioned in the discussion of the ætiology. An exception must be made as to diphtheria, which may be accompanied by either affection.

Pneumonia developing after prolonged submersion in water or fluid of any kind is quite certain to be of the variety under discussion.

After the disease has passed its primary stage, the difference between the two affections is more marked. Aspiration pneumonia runs its course as a rule within a week.

Prognosis.—The prognosis depends upon the nature of the associated condition or primary cause. It is most favorable in those cases which follow submersion in water. In other conditions of unconsciousness when the saliva and tracheal mucus enter the bronchi and alveoli the outlook is uncertain. In the newborn the disease is usually fatal.

Treatment.—The care of the patient must be on general lines, according to the individual demands of each case, which necessarily vary according to its cause. Inhalations of antiseptic vapors have been recommended, especially carbolic acid in a 4-per-cent. solution.

Prophylaxis is very important. Before administering an anæsthetic the mouth should be thoroughly cleansed with solutions of boracic acid, eucalyptus or similar non-toxic antiseptics. During coma or unconsciousness from any cause the mouth should be kept as aseptic as possible by the frequent applications of the same.

Should gangrene, abscess or empyema appear as complications, the treatment must be such as discussed in the consideration of those subjects.

PULMONARY TUBERCULOSIS.

SYNONYMS.—*Phthisis*, *Consumption*.

Pulmonary tuberculosis is an infectious disease caused by the tubercle bacillus, and characterized by breaking-down of the lung tissue, cough, purulent expectoration, sweats, and emaciation.

The widespread prevalence of pulmonary tuberculosis and the fact that more deaths are caused by it every year than by any other disease renders it one of the most important, and at the same time one of the most interesting, subjects with which the medical profession is confronted.

Ætiology.—Tuberculosis is found in all parts of the world, but is more general in the temperate zone.

Altitude also affects its development, for it is much less common in high altitudes.

Race exerts no little influence upon the development of the disease for reasons which are difficult to explain other than the fact that certain races seem to possess less inherent power to resist the inroads of the bacillus. In this country the Irish show the largest percentage of deaths from tuberculosis; the negroes the next largest, and the Jews least of all.

Tuberculosis affects the lower animals as well as man. It is common among bovines and fowls. In pigs it is also frequent; in sheep and horses rare. In monkeys and apes in the natural state it is unknown, but in captivity very frequent. In the cold-blooded animals it is seldom found even when in confinement. Avian tuberculosis presents different types from that of man, and it has been claimed that the bovine variety is not liable to infect man. This claim, which has been the subject of considerable discussion, however, has not been substantiated and the contrary holds.

The latest conclusions in regard to the bacillus of tuberculosis of man, the lower mammals and birds are: (1) That they are closely related and descended from one parent and that the different types have been developed by successive passages through individuals of a certain species, and by this process the bacillus acquired a virulence for certain animals, thus the bovine bacillus is more virulent for the lower animals than for man. (2) That certain morphological and bacteriological changes were also developed, which by suitable treatment can be made less divergent. (3) That bovine and human bacilli under favorable conditions are intercommunicable, but not to the extent formerly maintained. (4) That the milk from tuberculous cows is probably not so frequently a cause of tuberculosis in infants as has been for some time regarded, but it is none the less strenuously to be avoided, especially as it is the frequent source of fatal diarrhœa.

Bacteriology.—The direct specific cause is the bacillus tuberculosis, discovered by Koch in 1882. This micro-organism occurs in the form of fine, short rod-like bodies, slightly bent or curved, with an average length of about half the diameter of a red blood-corpuscle. In some instances there are lateral branches or outgrowths. Frequently the bacillus is found in pairs, closely adherent groups and strands. When once stained with fuchsin or gentian violet they are not discolorized by treatment with Gablet's solution or with a 20-per-cent. solution of any mineral acid followed by alcohol. The bacillus grows on blood-serum, glycerin-agar, bouillon or potato, but most readily on the first.

With reference to the type and arrangement of the tubercle bacilli as influencing diagnosis, Dr. H. P. Deady says, "I have observed that, generally speaking, a preponderance of thin short rods arranged or massed in groups are significant of an acute tubercular process either existing or approaching, and I have on different occasions been able to prognose the condition two to six weeks previous to the onset of subjective symptoms. It has also been noted that long rods irregularly arranged when in the majority are characteristic of early arrestment, while a greater number of the segmented rods are found in old chronic fibroids."

Experiments by Grancher and Ledoux-Lebard demonstrated that the bacillus from man grows after being immersed for fifteen minutes in water at a temperature of 122° F. A temperature of 212° F. caused sterilization. In a dry state the bacillus was shown to withstand a greater degree of heat. After seven hours' exposure to 150° F. it was virulent; also after three hours' exposure to 212° F. it was virulent. For thorough sterilization a temperature of 250° F. is necessary.

It has been proved that the dry sputum preserves its vitality in an ordinary apartment for about two and one-half months.

Koch discovered the important fact that the bacillus of tuberculosis when exposed to the direct rays of the sun was rendered sterile.

Streptococci and pneumococci are found associated with the bacillus tuberculosus of Koch in tubercular lesions. This has led to the view that the infection is multiple. However that may be, the primary cause is the tubercular bacillus.

Methods of Infection.—The means by which the bacillus enters the organism is by the mucous surfaces, food-inoculation and by carious teeth. The most common channel is by inhalation through the mucous surfaces. It is claimed that the expired air from a tuberculous patient is not infectious. The bacillus is contained only in the sputum, which when dry frees the organism and allows it to become widely disseminated in the atmosphere. In pulmonary tuberculosis the bacillus may gain entrance directly to the lungs by inhalation, or it may be conveyed there from a focus of infection from some other locality, such as the bronchial glands, pleura, tonsils, or by means of the lymphatics and venous circulation. There is a decided difference of opinion as to the channels of infection; some disputing infection by way of the bronchial surfaces, claiming the tonsils are the chief seat of entrance.

Another source of infection is by the use of meat and milk from tuberculous cattle. In the instance of meat from diseased animals, it has been shown that it is not always necessarily infectious. Moreover, when well cooked it will be rendered innocuous. With milk and butter infection is more likely to arise.

The direct introduction of the bacillus by inoculation may occur accidentally in many ways, as in wounds received in post-mortem examinations, cuts from a bit of infected glass or china, the rite of circumcision, wearing the clothes of an infected person, etc. The presence of carious teeth is a channel of infection and accounts in a certain proportion of cases for implication of the cervical glands.

Not every person who becomes infected develops the disease. The tubercle may find a nidus, but the soil, so to speak, not being favorable, it becomes encapsulated and its course totally arrested. This is illustrated by the finding of tubercular deposits in those in whom there was no evidence of the disease during life.

CONDITIONS FAVORING INFECTION.—It is well recognized that the disease is much more prevalent in thickly populated localities than in the country. The influence of sunlight has been mentioned. Experiments show that infection spreads with a much greater degree of virulence in its absence. In brief, it may be said that in proportion to the deprivation of fresh air and sunlight the bacillus of tuberculosis flourishes. Soil is also a factor; if wet and badly drained it favors the development of the disease.

The predisposing influences intimately associated with the patient individually are important. A phthisical diathesis has been recognized since the days of the early fathers of medicine. This is generally understood to be a slender physique, flat chest, long slender bones, flaring scapulæ, clear, transparent skin, bright eyes and oval face. This type simply indicates a delicate constitution and feeble resistive powers. Tuberculosis, however, is not confined to individuals of this type, for it frequently makes its appearance in persons with broad and well-developed chests, especially among the lower classes. In these instances a naturally strong physique falls beneath bad hygiene and alcoholic excesses.

Heredity exerts a strong influence. The disease may be directly transmitted, as shown by post-mortem examination, but this is rare. The usual manifestation of heredity is the transmission of a diathesis or constitution favorable for the development of the disease. This influence is observed in a large proportion of cases.

The disease is much more prevalent among males than among females, at least in the city of New York, the proportion being about three to one. This statement is in direct contradiction to that of other writers. It is based upon the statistics of the Tuberculosis Infirmary of the Metropolitan Hospital.

The influence of race has already been mentioned.

As far as age is concerned, while there is no period in life in which the disease may not occur, it is rare in infancy and childhood and comparatively rare in old age, being more generally prevalent between the eighteenth and fortieth year. Recent investigations show that pulmonary tuberculosis is more frequent among the aged than was once supposed, but it is usually latent or very slow in progress. It was also formerly maintained that the disease never occurred in an emphysematous patient. This also has been shown to be without foundation and the association of the two lesions, while rare, is not so much so as was formerly maintained.

Occupation and habits of life are important factors. Tuberculosis is frequent among workers in mills where the atmosphere is laden with dust and other impurities and among glass-blowers, miners, stone-cutters, laborers in excavations, etc. Among people engaged in occupations of this nature over-indulgence in alcoholic stimulants and those of a very inferior grade, poor food and bad hygiene are common contributing causes.

In addition to general causes there are many local morbid conditions which pave the way for the lodgment and development of the bacillus. In many instances the association of the predisposing and general influences will be very apparent, while in others it may not be so. The most important of local morbid conditions which actively tend to the affection are as follows:

Bronchitis is one of the most frequent forerunners of pulmonary tuberculosis. The inflammatory process which involves the bronchial mucosa seems in some way to enable the bacillus to enter the system. An extensive pleurisy with effusion is frequently followed by the disease. The prolonged compression to which the lung is subjected and the change in its structure which follows seem peculiarly fitted to prepare the way for the infection.

Influenza, whooping-cough and measles, on account of the associated bronchial complications, are sometimes followed by pulmonary tuberculosis. Diabetics, as is well known, are especially prone to the disease, and a large proportion of cases terminate in this way. Any long-continued illness, such as typhoid, or heavy drain upon the system, such as rapid child-bearing and prolonged lactation, renders the system susceptible to the inroads of the micro-organism, especially in those in whom there is an inherited tendency.

Persons with contraction of the pulmonary artery, congenital or acquired, generally die of pulmonary tuberculosis. The relation of valvular diseases of the heart to pulmonary tuberculosis is not so clear. It is claimed that they especially favor its development. A congenitally small heart is supposed to predispose to the disease.

Trauma is a causal factor. Blows upon the chest or fracture of the ribs may contuse or otherwise injure the lung tissue and form the focus of infection.

General Histology.—In its incipiency, tuberculosis may be regarded as a local disease dependent upon the presence of the tubercle bacilli at some given point from which, as a focus, infection spreads to other parts or becomes general. When the bacilli enter through the lymphatic system they develop and proliferate near the seat of entrance. When through the blood they become scattered throughout the lungs. When through the lungs by inhalation they lodge in the bronchi or alveoli.

The bacillus, having obtained lodgment, develops and multiplies and gives rise to the miliary tubercles which are the direct product of the toxins and histologically the essential primary element in tuberculosis. The tubercle in appearance, as viewed by the naked eye, is a grayish, bead-like body. Microscopically its component parts are seen to consist of the bacillus, epithelioid cells, leucocytes, giant cells and a reticulum. The bacilli are found in both epithelioid and giant cells, in a free state between them and in all tuberculous tissues. The epithelioid cells are the results of proliferation of the fixed tissue cells, especially those of the connective tissue and the epithelia of the capillaries. They are rounded, cuboidal and polygonal bodies with vesicular nuclei, inside of some of which the bacillus is seen.

Giant cells are found in some, but not all tubercles. It is claimed that they are the product of the increase of protoplasm or nuclei of an individual cell or of fusion of individual cells. This view is not endorsed by some observers who maintain they are the result of cell growth without division or fusion of several cells. The giant cells contain many nuclei.

The leucocytes appear by migration from the vessels of the affected area and accumulate in the margin of the focus of infection. These cells do not subdivide, but increase by aggregation of the so-called wandering element. According to Metschnikoff, the leucocytes oppose the bacilli and under favorable conditions destroy them.

The reticulum originates in the connective tissue matrix by fibrillation and rarefaction. As the reticulum develops the margin of the tubercle becomes more pronounced and as the result of pressure within, the blood-supply gradually becomes diminished and the tubercle undergoes degeneration. This may appear in two general varieties, the caseous and the fibrinous. The former is disintegrative and destructive; the latter conservative. The power of the organism to resist the bacillus determines the nature of the process which ultimately develops. In the instance of caseous degeneration these powers are feeble. The cellular elements of the tubercles undergo coagulation and necrosis, losing their outline, their ability to take stain, and become a structureless mass which in time undergoes fatty degeneration. Tubercles undergoing caseous degeneration in aggregation form the cheesy masses usually observed in the lungs. The mass may soften and rupture, or it may become encapsulated and undergo calcareous change.

In the fibroid form of degeneration there is an overgrowth of connective tissue which changes the tubercle into a hard mass. Frequently the change is of a fibroid caseous nature, with the former predominating. This condition is more common in the peritoneum than elsewhere.

When the tubercle undergoes caseous degeneration and encapsulation does not take place, there is a marked tendency on the part of the infection to spread. When, on the other hand, the process is limited by encapsulation, dissemination of the infection is prevented. In these instances the bacilli may sometimes remain latent for years, but at any time the capsule may rupture from the invasion of streptococci, and the infection become general.

Infection from tubercular nodules may be disseminated in various ways. Particles of a nodule situated in the bronchial mucous membrane may become detached and carried into the bronchioles by the respiratory act and thus establish new areas of infection. Again, when the nodule is seated in the lung structure, bacilli may be carried into other parts of the lung through the lymphatic circulation by means of the phagocytes which migrate, carrying with them the bacilli, then die, leaving the latter distributed in new localities. Thus different parts of the organism become affected.

According to the mode of infection there are two general types of lesions, namely:

(1) When the bacilli enter the lungs through the blood-vessels or lymphatics, the morbid process first appears in the alveolar walls, in the capillaries, in the epithelia of the air-cells and in the connective tissue of the septa. The irritation of the bacilli within a few days may develop the tubercles as mentioned, which may be confined to one lung, scattered throughout both, or the process may form part of a general tuberculous infection.

(2) When the means of entrance is by inspiration, the smaller bronchi and bronchioles are more extensively involved. The process is more lobular and the tuberculous deposits larger, more general, and in some instances involve the greater portion of one lung. It is under these conditions that peri-bronchitis and nodular broncho-pneumonia appear.

Three forms of pulmonary tuberculosis are generally recognized, the acute, the chronic ulcerative and the fibroid.

1. Acute Pulmonary Tuberculosis.

This variety occurs in two forms, the pneumonic and the broncho-pneumonic. Either may arise primarily in a previously healthy person or secondarily from some pre-existing infection or follow some debilitating disease.

Acute Pneumonic Tuberculosis.

Morbid Anatomy.—In the pneumonic form usually one lobe is implicated, but the entire lung may be involved. The affected portion on examination is seen to be deeply congested, heavy and airless. The pleura is covered with a thin exudate. On section the surface is seen to be much congested and studded with grayish-white nodules the size of a shot. These nodules are gelatinous and transparent. They are the tubercles, the aggregation of which give the lung a variegated appearance. It is generally grayish in color with deeply congested tissue between the tubercles. There may be areas of yellowish-white caseous masses of degeneration when the process has been of longer duration. Small cavities with irregular walls are found scattered throughout the involved areas.

Pneumococci may be present, but are not an active factor. The process usually begins in the upper lobe. It belongs to adult life and is seldom observed in children.

Symptoms.—The disease is usually ushered in abruptly with a chill, although there is frequently the history of exposure to some debilitating influence. The chill is quickly followed by a rapid rise of temperature, the thermometer registering 104° F. to 105° F. The fever is continuous with remissions of 1.5° to 2°. There may be severe pain in the side from pleuritic complications. There is cough with expectoration, which at first is mucoid and later rusty colored and muco-purulent. Examination shows the bacilli. Sometimes there is hæmoptysis. Dyspnœa is pronounced and the respiration rapid and superficial. There may be attacks of suffocation. Night-sweats are present as a rule at the end of the first week. They are usually excessive and exhausting. Emaciation is rapid and prostration extreme. Instead of a crisis about the eighth day the condition becomes more serious, the temperature irregular and pulse more rapid. The perspiration now becomes more profuse and the expectoration muco-purulent and greenish in color. As emaciation and prostration increase, the signs of breaking down of the lung tissue become evident.

Mental symptoms are absent and the patient may appear quite cheerful even up to the last stage of the disease.

PHYSICAL SIGNS.—The signs are those of consolidation; feeble or suspended vesicular murmur and later well-defined bronchial breathing.

Diagnosis.—The similarity of acute pneumonic tuberculosis at the onset to croupous pneumonia is very striking. The principal features by which the tuberculous process may be distinguished is the more usual implication of the apices, profuse sweats and remittancy of the fever. During the first week, however, it is often difficult to clearly differentiate the two conditions, but after that much less so. The absence of crisis and the course of the fever are distinctive. Examination of the sputum and the detection of the bacillus of tuberculosis and shreds of elastic tissue will enable the diagnosis to be made at any period.

Prognosis.—Death may occur in the second or third week or the patient may linger for two or three months. In some instances the disease may pass into the chronic form.

Broncho-pneumonic Tuberculosis.

This is a more common type and is that which is known as "galloping consumption" or "phthisis florida." It is more frequent among young adults and in children.

Morbid Anatomy.—The disease is an acute broncho-pneumonia characterized by the presence of caseous matter. It usually involves both apices from which it extends downward or it may be scattered throughout different portions of the lung. The process begins in the smaller tubes which become filled with the caseous matter, while the alveoli in turn become filled with the products of inflammation. Thus there will be solidification of the lobules. In some instances an aggregation of these lobules by continuity and fusion form a solid mass which may involve the whole or the greater portion of the lobe. Generally, however, consolidation of the lobules is observed with crepitating tissue intervening.

In some instances broncho-pneumonic tuberculosis first appears after hæmorrhage. The blood settles in the alveoli and bronchioles spreading the foci of infection and the process develops. The hæmorrhage may occur in persons in whom no lesion had been previously suspected, or the process may follow hæmorrhage in cases where the chronic form of the disease had been previously well recognized. This type is sometimes called tuberculous aspiration pneumonia. On section the lung shows grayish-white areas of consolidation with crepitant tissue intervening. In some instances there are cheesy masses of varying size, some as large as a cherry. The cheesy matter softens, breaks down and leaves cavities with ragged walls. Between these cheesy masses, especially in the lower lobe, there may be areas of recent pneumonia, the lung generally undergoing hepatization. In children enlarged bronchial glands may be observed at the root of the lungs which may pass directly into their structure. The lobules under these conditions may be involved by direct contact.

Symptoms.—The appearance of the disease is variable. It may attack those who are in apparent good health, but who are temporarily run down or debilitated from any cause. The initial symptoms in some cases are repeated chills with high temperature, quick pulse, dyspnœa, and rapidly progressive emaciation and prostration.

As previously mentioned, hæmorrhage may be the first symptom; when such is the case the disease may run a very rapid course. In such instances it is to be presumed that a latent unrecognized focus of infection was present.

In children the disease may follow the acute infections, more especially measles and whooping-cough. Under these conditions it may manifest itself in several ways, viz., with cough, dyspnœa, high fever and the signs of consolidation, death following in a few days; with the symptoms such as ordinarily attend a severe grade of broncho-pneumonia, death occurring about the sixth week; or with the more gradual development of symptoms peculiar to tuberculosis during convalescence from some acute infection, the condition eventually assuming the chronic form of pulmonary tuberculosis.

In all types of the disease the fever runs generally high, but is irregular in its course with frequent remissions, as illustrated in Fig. 46. Cough appears early and is constant. Expectoration at first is mucoid, later muco-purulent and bloody. Microscopical examination shows the presence of bacilli and fibres of lung tissue. Perspiration is excessive and exhausting and emaciation rapidly progressive.

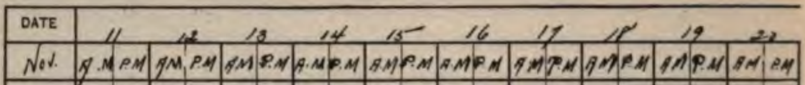

FIG. 46.—Temperature chart of a case of acute pulmonary tuberculosis. First manifestations of symptoms five weeks previous, night-sweats, loss of flesh, weakness and cough marked. There was also some hæmorrhage. Both apices were involved, showing the presence of consolidation and râles. Patient, male, aged 41 years. Flower Hospital.

In some cases after the disease has run for about three weeks, the patient sinks into a typhoid state with delirium, dry, coated tongue, and high fever. In others the course is more protracted. For six or eight weeks there may be excessive sweats, chills, fever, cough, muco-purulent expectoration, rapid emaciation and signs of pulmonary consolidation, when the intensity of the symptoms may subside and the course of the disease assume a chronic form.

PHYSICAL SIGNS.—In the early stages the signs are the moist râles of bronchitis. These are soon accompanied by areas of dulness on percussion with broncho-vesicular and bronchial respiration and subcrepitant râles. Later when softening occurs, signs denoting the presence of cavities develop. The morbid processes are usually, as stated, in the apices and are bilateral.

Diagnosis.—The excessive sweats, rapid emaciation, high fever with irregular remissions and hæmorrhages indicate that some disease other than an ordinary broncho-pneumonia is present. The conclusive test is the detection of the tubercle bacilli and elastic-tissue fibres in the sputum.

Prognosis.—It is needless to state that the prognosis is very unfavorable. A few cases, however, recover.

Treatment.—In both forms of pneumonic tuberculosis remedies appear to have little effect. The strength of the patient should be sustained by the most nutritious diet. The fever when excessive may be controlled by cold sponging and baptisia. Arsenici iodidum, phosphorus, baptisia, iodinum, ferrum phos. and antimonii iodidum may be administered as indicated.

2. Chronic Ulcerative Tuberculosis.

Under this head may be grouped the larger proportion of cases of pulmonary tuberculosis. While the process is primarily and essentially tuberculous, symptoms ultimately develop which indicate sepsis from the absorption of purulent matter incident to the breaking down of the lung tissue.

Morbid Anatomy.—The first morbid process after infection is the formation of the tubercles. In the great majority of instances this begins near the apex in connection with a bronchiole or air-cell. Sometimes the process is unilateral, sometimes bilateral. In the latter instance generally one lung is more deeply implicated than the other. The base of the lungs are most free from morbid changes, especially on the side opposite that in which the apex is more markedly involved. In instances where the process is said to be unilateral, the lung on the other side shows some changes.

Tubercular infiltration usually commences about one or one and a half inches below the upper margin of the apex near its distal and posterior borders. From thence it spreads downward. Frequently the first evidences of consolidation can be detected posteriorly in the supra-spinous fossa. Anteriorly the primary area of infection corresponds to a point below the centre of the clavicle. The process gradually extends along the upper lobe anteriorly, running in a line about half an inch from the proximal ends of the first, second and third intercostal spaces. Less frequently the primary area of involvement is situated below the outer third of the clavicle. In this instance the downward extension of the process is along the outer border of the lobe. Implication of the middle lobe usually follows disease of the upper on the same side, the process extending in the same line. In the lower lobes the same tendency for the disease to first appear in the upper portion is also manifest. The primary area of involvement in this instance is at a point an inch and a half below the posterior extremity of the apex (of the lower lobe) corresponding in the chest to a point on a line with the fifth dorsal spine. From this situation the process extends downward along the posterior margin of the lung and laterally along the interlobular septum. On the chest wall it corresponds to the vertebral border of the scapula when the arm is raised behind the head.

Involvement of the lower lobe on the affected side is of important significance and is often manifest before there is any marked progress in the apex of the other side.

It is apparent that communication with the veins or bronchi by ulceration greatly facilitates the spread of the infection, which is comparatively slow when limited to the lymphatics. A possible change which may follow the discharge of a tubercle into the systemic circulation is the spread of the infection to all parts of the system and a consequent acute miliary tuberculosis. Primary lesions of the base are very rare.

Ultimately a variety of morbid changes follow the presence of the miliary tubercle, namely, areas of consolidation, nodular tuberculous masses, caseous masses, suppuration, sepsis, sclerosis, implication of the pleura and bronchial glands, and general tubercular infiltration.

Consolidation of areas of the lung is an essential part of the morbid process in pulmonary tuberculosis. It begins at the apex, as a rule, and follows the line of the tubercular infiltration as mentioned.

In the great majority of cases the process is a broncho-pneumo-

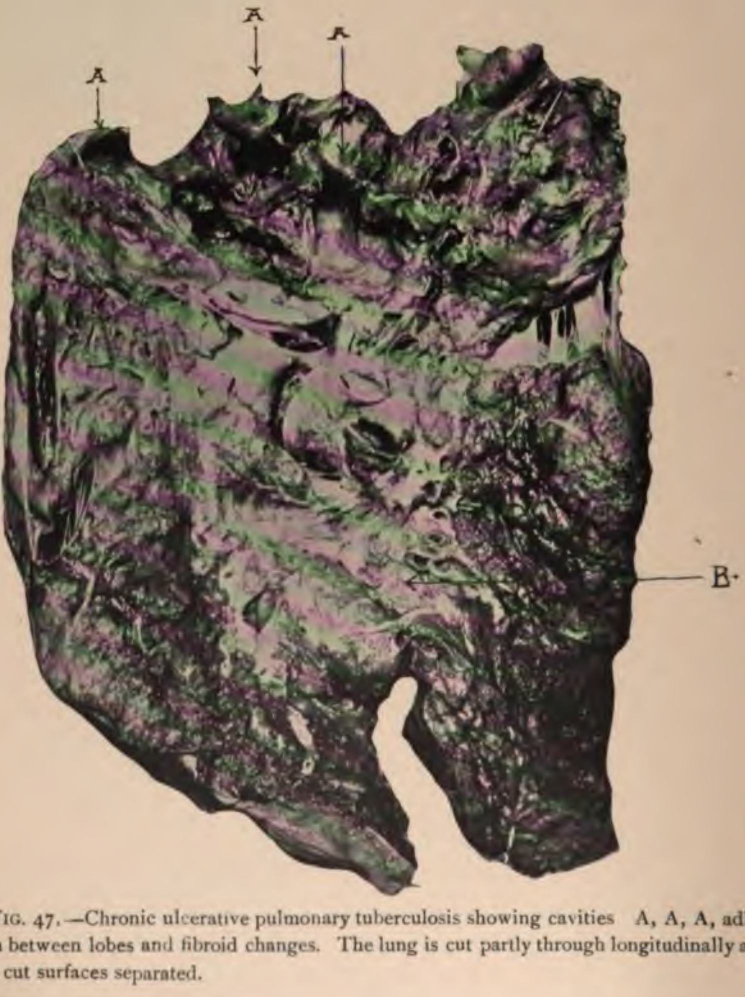

FIG. 47.—Chronic ulcerative pulmonary tuberculosis showing cavities A, A, A, adhesion between lobes and fibroid changes. The lung is cut partly through longitudinally and the cut surfaces separated.

nia. Examination discloses the seat of inflammation in the terminal bronchioles. The steady growth of the primary tubercle causes it to encroach upon the alveolar tissues, whence a catarrhal inflammation leads to consolidation from the accumulation of the exudate in the air-cells. Associated alveoli become involved and likewise filled with the products of inflammation which is found in all stages of caseation.

On section of a broncho-pneumonic patch the bronchiole is seen in the central portion filled with the products of inflammation in a high degree of degeneration which involves the walls of the alveoli. Surrounding this is a zone in which the alveoli are filled with epithelial cells which have undergone more or less fatty and granular degeneration, while externally to all there is still another zone in which there is little or no degeneration in the inflammatory products. As the areas of broncho-pneumonia continue to develop, there is more or less tendency on the part of those in close proximity to fuse and form large masses of consolidation.

A secondary but very important feature is the pneumonic inflammation which arises in the alveoli surrounding the tubercles. When the tubercles are very close together the intermediate contiguous tissues undergo a catarrhal inflammation and in consequence become filled with masses of exudate in the form of epithelia, small round cells and fibrin. The result of this process is large consolidated areas which upon section present a grayish appearance, with yellowish or yellowish-gray tubercles scattered throughout. Some writers describe a red hepatization as occasionally observed.

The next series of changes are those which occur in the consolidated areas. These are ulceration, caseation and softening, excavation and sclerosis. The infiltrated tuberculous areas undergo a caseous metamorphosis which is a coagulation necrosis. The caseous mass is yellowish and cheesy. Closely contiguous tubercles may fuse together and form large cheesy masses, sometimes crumbly, soft and purulent. This substance can never undergo resolution, but it can be cast off through the bronchi or become encysted or calcified. Fatty metamorphosis also occurs.

The cheesy masses undergo softening and are discharged from their seats and thus cavities are formed. Cavities are the source of perpetual auto-infection. They are the direct result of ulceration and necrosis and are essentially different from those of bronchiectasic formation. Their starting-point in most cases is in the walls of the bronchi in a tuberculous area, dilatation of which frequently results from retention of secretion and ulceration, and necrosis of the walls follows with more or less destruction of surrounding tissue. As the disease advances the cavity becomes larger. New ones form and contiguous ones coalesce. Thus there may be a series. In most instances there is communication with a bronchiole as stated, although in some cases it is otherwise. Associated with the necrotic process there is more or less sepsis arising from secondary infection by the staphylococcus pyogenes.

Cavities are generally situated in the upper lobe near the apex. They are not as a rule large, but in exceptional instances may be very extensive. Their surfaces may be smooth, with a well-defined lining membrane or ragged and rough without distinctive lining. Sometimes the lining membrane is well supplied with blood-vessels and may show a decided tendency to hæmorrhage. The walls are often crossed by trabeculæ which represent former blood-vessels and bronchi. Their contents are usually purulent, similar in character to that expectorated. Occasionally they are very foul, especially when the walls are gangrenous, as they sometimes are.

The change which yet remains to be discussed is that of sclerosis. This is a conservative effort of nature. It is seen in cases which run a slow course where the process of softening has not advanced very far. It consists of the formation of fibrous tissue in the involved areas, which may be limited to the margin of the tubercular mass which it encapsulates. Within the fibrous capsule the tubercle may remain a cheesy mass and at some period later the capsule may ulcerate from streptococcus invasion and liberate the bacilli, or the tuberculous mass may undergo calcification which may, together with sclerosis, be said to represent healing of the process. In this instance, also, the capsule may ulcerate and calcareous masses may be expelled. These are the so-called lung-stones.

An important change in association with the encapsulation of the tubercles is the fibroid induration which develops in the intervening tissues. It is the result of the inflammatory process primarily induced by the bacillus and is conservative in its tendencies.

Thus in cases of arrested pulmonary tuberculosis, areas of fibroid induration of the lung are most always observed.

The pleura in nearly all instances is involved. Adhesions are almost always found, especially near the apex. They may be friable or dense and firm. In some instances they are extensive. The pleuritic process is usually simple, but it may be distinctly tubercular. Effusion may occur, either serous, purulent or hæmorrhagic. Pneumo-thorax from rupture of the walls of a cavity near the surface may likewise result.

Changes in the bronchi constitute an important element in the pathological process. The large tubes are more or less in a condition of chronic inflammation, the result of extension upward from the areas of consolidation when the bronchioles are first involved. The mucosa of the tubes is congested and roughened and ulcerated in patches. The peri-bronchial connective tissue is also inflamed and thickened. Obstruction of the tubes occurs and leads to scattered areas of atalectasis, or the walls of the tubes may give way and dilate and lead to bronchiectasic dilatation.

The bronchial glands are generally enlarged, indurated, pigmented and contain caseous, purulent points or foci.

It is evident, considering the nature of the disease in question, that infection of other organs may be expected in association with that of the lung. The most frequent is the larynx and intestines. The liver, kidney, spleen, meninges and serous membranes generally may also show evidence of tubercular complications.

Changes in the heart are not especially characteristic. In some instances it is small, either congenitally or from atrophy. Fatty infiltration is comparatively common. Fatty degeneration may also occur.

Symptoms.—The symptoms which attend the onset of chronic ulcerative pulmonary tuberculosis appear in a variety of forms, some vague and insidious, some strikingly characteristic, while others tend to divert attention from the actual condition. The initial or early symptoms, therefore, are best considered from the standpoint of certain types, the chief of which are as follows:

(1) In by far the greater proportion of cases, the symptoms of a bronchitis first attract attention—"a neglected cold" is a common expression. The patient notices he has a cough which is more or

less persistent and which is usually attended with expectoration, although it sometimes may be dry and hacking.

In cases of this type sometimes the disease may make great advances before the patient's attention is aroused. This is not, however, because of latency or insidiousness of onset, but because through ignorance the patient fails to recognize the condition, persisting in thinking he has only a "cold." Such cases are more frequent among the lower classes and are often observed in public institutions. For example, a large proportion of those admitted to the Metropolitan Tuberculosis Infirmary on Blackwell's Island are far advanced in the disease, yet never before have sought relief.

(2) There is a considerable group where the onset of the disease is exceedingly insidious and where in the early periods it remains apparently latent. In such instances the process may make decided inroads before the patient is aware that anything serious is the matter.

(3) Hæmoptysis is not infrequently the first symptom. In most of such cases a tuberculous infection is present, but so latent that the patient may appear in good health at the time. The hæmorrhage may be soon followed by other pulmonary symptoms, or there may be an interval of several months when there is a recurrence, which is shortly followed by the development of decided evidences of lung disease.

(4) Pleurisy of the dry variety at the apex may be the first noticeable symptom in some cases. Pleurisy with effusion in many instances is likely to be followed by pulmonary tuberculosis, especially when the effusion is apparently sterile.

(5) In some cases the symptoms may assume the appearance of malarial fever. There are paroxysms of chills, fever and sweats, occurring with regularity. In localities where malaria is prevalent great care must be exercised to avoid error.

(6) Gastro-intestinal symptoms and anæmia introduce pulmonary tuberculosis in another group of cases, and may tend to divert attention from the real cause. In such cases there is gastric irritability, nausea and acid eructations and vomiting. In young persons, especially in girls, in association with these symptoms there may be marked anæmia.

(7) Anæmia without decided gastric symptoms other than ano-

rexia, but with prostration, palpitation, slight fever in the afternoon, is another type of initial symptoms. This group also includes anorexia with a progressive cough and loss of weight.

(8) Laryngeal symptoms are the first manifestations in a number of cases. In many of these the disease is limited, as far as can be recognized, to the larynx, but it is probable there are latent foci of infection in the lungs as well.

(9) Tuberculosis of the cervical and axillary glands is another source of infection and one which may be considered as an initial group. In these cases there may be enlargement of the cervical and axillary glands for many months before the manifestation of pulmonary infection. The importance of the removal of tuberculous glands cannot be too forcibly emphasized ; also carious teeth.

(10) Persistent high pulse in association with symptoms of prostration may be considered as an early symptom suggesting tuberculosis.

(11) Anal fissure and chronic diarrhœa, especially if of long standing, should always suggest the possibility of tubercular infection.

In a typical case of chronic ulcerative pulmonary tuberculosis, the principal symptoms are cough, expectoration, chest pains, fever, night-sweats, gastro-intestinal disturbances, dyspnœa, general weakness, prostration and emaciation. The course is variable and the manifestations of symptoms during the stages of formation of the tubercles, softening and formation of cavities not always constantly correlative. The symptoms therefore will be considered individually and not necessarily in sequence.

Cough.—This is an early symptom and one which in a very large proportion of cases is the first to arouse attention. It is also almost always present, although there are exceptional cases where the patient claims he has not had any cough. In many such cases, however, it will be found that there has been a slight cough which the patient did not take into consideration. At first the cough is dry and hacking. Later it is moist and as the disease advances severe and harassing. When cavities form it may be paroxysmal. Nocturnal exacerbations are a marked feature, especially in the advanced stages when they cause great distress and loss of sleep. In some instances cough is attended with vomiting and by the continual loss of food may contribute very greatly to the emaciation.

Expectoration.—This is an almost constant symptom, yet occasionally instances are observed when notwithstanding the presence of well marked consolidation, hectic and slight cough, it is absent. Ordinarily in the early stages expectoration is scanty, chiefly catarrhal, clear and viscid. Later, as softening progresses, it becomes much more abundant and contains small gray or greenish purulent masses. Later still, it becomes more purulent. In some cases the purulency predominates to a marked degree. The quantity may amount to as much as eight or ten ounces a day. A feature of the amount of sputum is its greater abundance at certain times in the twenty-four hours, mainly in the morning on waking and usually also in the afternoon. The patient may make no mention of its taste, or he may say it is salty or sweetish. There may be a faint, sickish odor or there may be decided putridity. The latter denotes the presence of bronchiectasis or cavities. When small muco-purulent masses appear the presence of tuberculosis may be suspected, for it is not until then that the sputum may in any way be said to be characteristic. These masses should always be examined microscopically. With the formation of the cavities the sputum assumes what is called the nummular character, that is, each of the greenish masses is airless, flattened and falls to the bottom of a vessel when placed in water.

Microscopical examination is a matter of the utmost importance. The typical case show mucus, tubercle bacilli, cheesy matter, pus, elastic fibres and in some instances blood. The evidence of the tubercle bacilli in the sputum is conclusive proof of pulmonary tuberculosis.

One of the best methods of staining for examination is the Erhlich-Weigert, which is as follows: Add eleven centimeters of a saturated solution of fuchsin in absolute alcohol to one hundred centimeters of a saturated solution of commercial aniline oil; this is to be prepared by shaking up the oil in water and then filtering, and should be freshly prepared every third or fourth day. A small piece of the sputum mass is placed by means of a needle or wire on the top cover and spread out so as to make the layer as uniformly thin as possible. The top cover should then be slowly dried, holding it about a foot above a Bunsen burner for about a minute. Some of the staining solution is then dropped upon the top cover which is still held above the Bunsen burner until the solution boils. The staining solution is then washed away with distilled water, decolorized in a 20 per cent. solution of nitric acid, washed again and mounted on the slide. The appearance of the bacillus has already been described.

Elastic tissue may come from the bronchi, the alveoli or the coatings of the arteries. It signifies breaking down of the lung tissues and is not confined to pulmonary tuberculosis. Examination may be made as follows: Spread a thin layer of the mass of elastic tissue on a glass about three inches square, flatten this by pressing on another glass. On a black background the fragments of elastic tissue may be seen by the naked eye as greenish-yellowish points; or they may be placed under a low power microscope and more readily distinguished. Another method is to boil some of the sputum for a few minutes in an equal quantity of a solution of caustic soda in the proportion of twenty grains to the ounce. Let the fluid stand for twenty-four hours in a conical glass, then examine under the microscope. The tissue from the bronchi will be observed as elongated network, or may appear as several long fibres bound together. That from the blood-vessels is somewhat similar; sometimes a small portion of the intima may be seen. Fibres from the alveoli are branching and may retain the outline of the alveolus from which they were detached. Calcareous masses as mentioned ("lung stones") in the sputum are sometimes observed. The size of these bodies varies from a small pea to a cherry. Generally but one is ejected at a time. Their origin is due to a calcification of the cheesy matter. They may also come from the bronchial gland, from which they penetrate into the bronchial tubes by ulceration.

Chest Pains.—These are a very common symptom. The usual seat is the supra- and infra-clavicular, the mammary and lower axillary regions. They are usually due to dry pleurisy or the stretching of old adhesions. In some instances they may be due to a neuritis of the intercostal nerves.

Respiration.—The breathing is always more or less quickened. Generally speaking, the greater the amount of lung tissue involved the higher the rate of respiration. Increase is especially noticeable in the evening and on exertion. The patient, however, does not complain much of shortness of breath unless in the latter stages, and even then the respirations may be very markedly increased without great discomfort. The absence of dyspnœa under these circumstances is attributed to the reduction of the volume of the blood, the general anæmia and the loss of tissue bulk.

Dyspnœa may be urgent in the last stages, after severe paroxysms of coughing and when there are extensive pleuritic adhesions, larger effusions into the pleural cavity, and extensive and rapidly-forming consolidations.

The Pulse.—The rate of the pulse is accelerated and its volume diminished. A quickened pulse, together with the symptoms pointing to tuberculosis, is of significance. In the earlier stages it ranges from eighty-five to one hundred; later from one hundred to one hundred and twenty, according to the degree of fever.

Fever.—The type of fever may be either intermittent or remittent. Either may occur at any period of the disease, or the two types may alternate. The maximum degree of temperature is usually reached between 2 and 6 P.M., and the minimum between 2 and 6 A.M. This feature should not be overlooked. An erroneous idea would result if the temperature is only taken at 8 or 9 A.M. and 8 or 9 P.M. When intermittent in character it is liable to be subnormal in the morning. A typical range of temperature is normal or subnormal in the morning, a gradual rise to 102° to 104° F. in the early evening or afternoon, then a gradual fall to normal or subnormal. This gives quite an even temperature curve. Fever signifies the presence of the inflammatory process which results from the irritation of the bacillus, the breaking down and suppuration of the lung tissue and the absorption of septic matter, that is, a secondary infection. The actions of these influences, except perhaps in the early stages, are difficult to separate, as they operate conjointly.

In some instances there is chilliness preceding the rise of temperature. Attention has already been called to the possibility of error in mistaking such cases in the earlier stages for malaria.

The higher the range of temperature and the greater its persistence the more active the process. In the earlier periods, during the formation of tubercles, and when softening begins fever is nearly always present. In the latter stages it is generally remittent in type, falling one or two degrees in the morning.

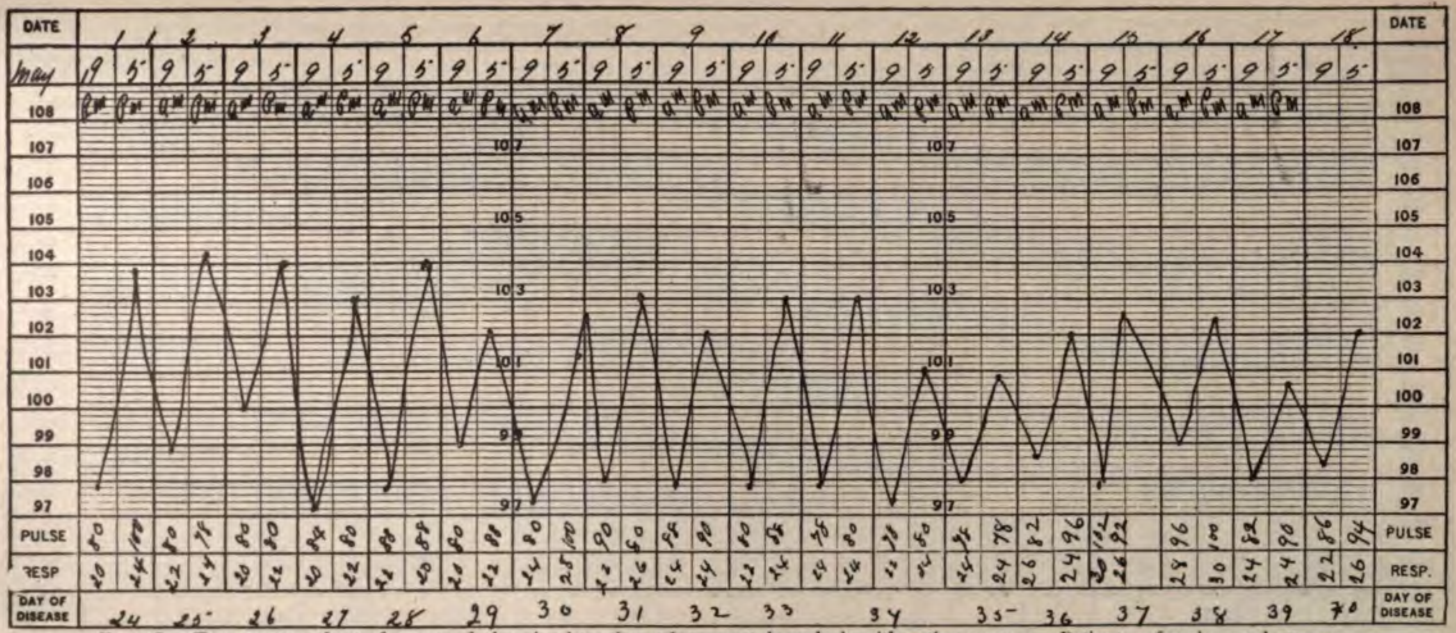

FIG. 48.—Temperature chart of a case of chronic ulcerative pulmonary tuberculosis with active process. Patient, a female, aged 47 years. Metropolitan Hospital.

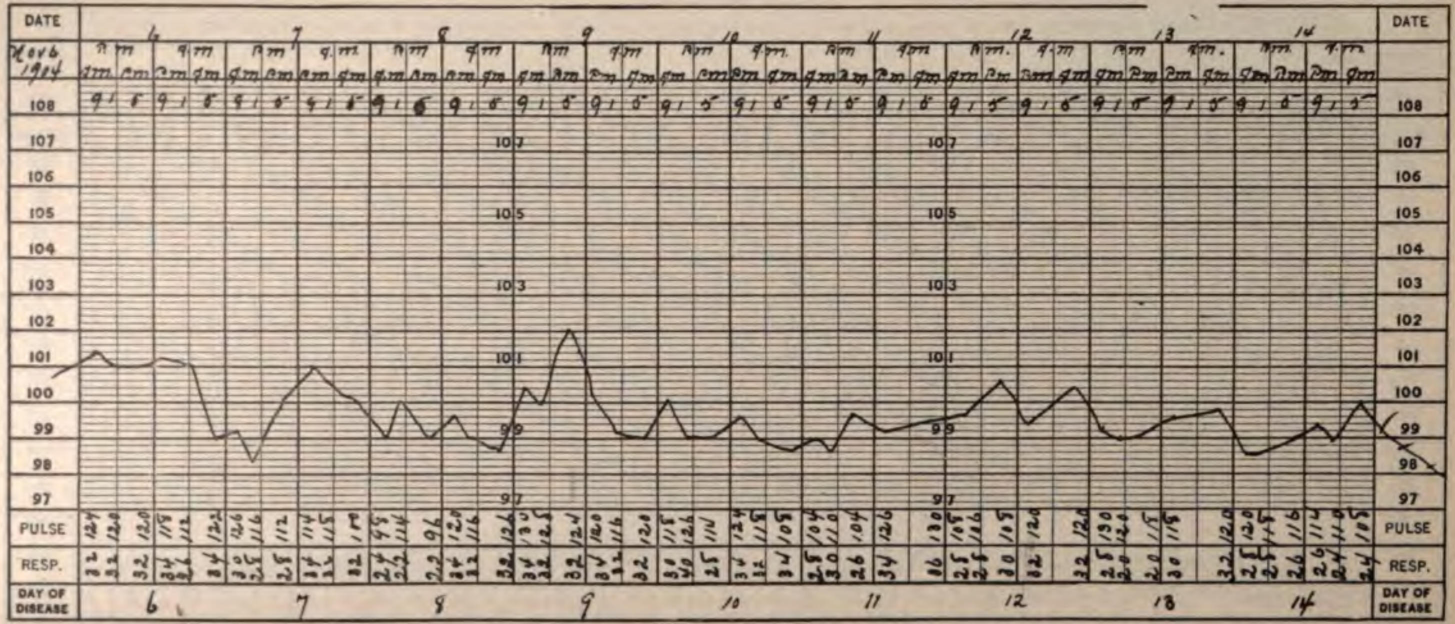

FIG. 49.—Temperature chart of a case of chronic pulmonary tuberculosis. Patient, a male, aged 29 years. Metropolitan Hospital.

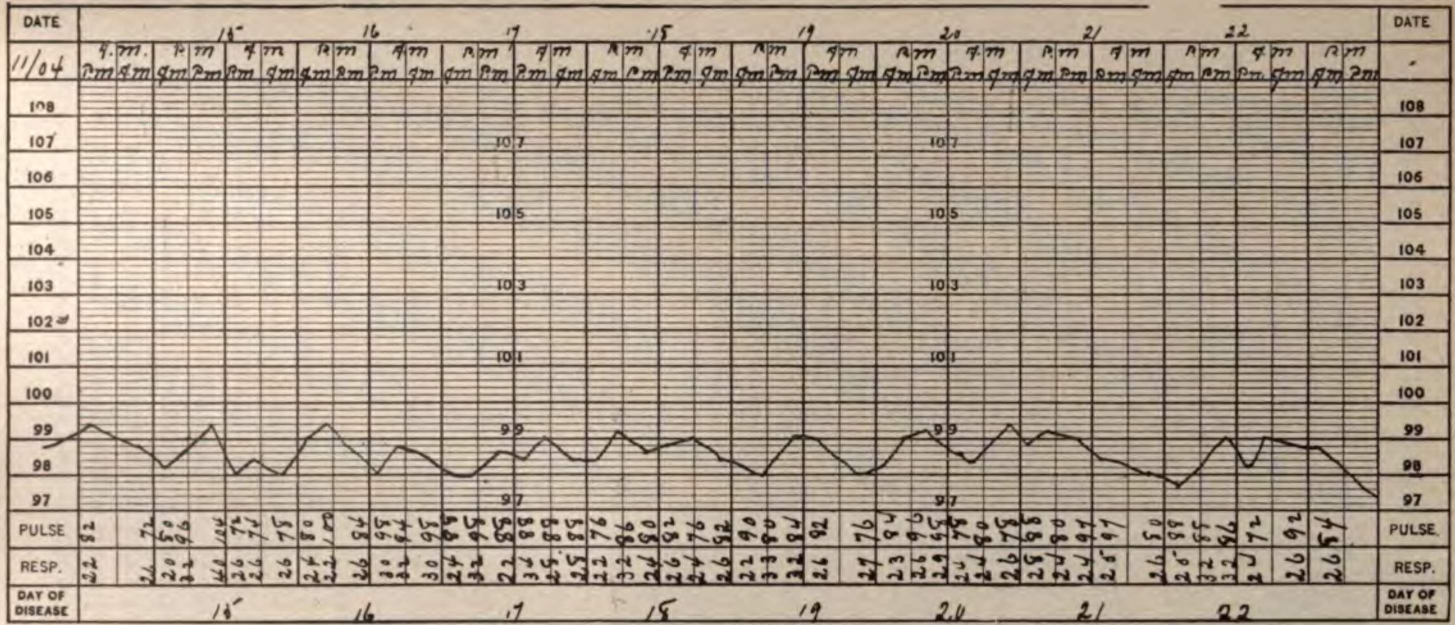

FIG. 50.—Temperature chart of a case of chronic pulmonary tuberculosis. Patient, a male, aged 20. Metropolitan Hospital.

It should be borne in mind that many cases are without fever during a certain portion of the day. The more chronic the type of the disease and the slower its process the more likely are the afebrile periods to be observed. In exceptional cases marked changes in the lung may be present without fever.

When the temperature ranges high and there are slight remissions, the presence of an acute pneumonic tubercular process should be suspected.

Sweats.—These are a common symptom and one which is the source of much distress and exhaustion. They may appear early in the disease, but are more frequent when it has progressed to the stage of breaking down of the lung and cavity formation. They are most likely to occur in the early morning after the disappearance of the fever, or at any time when the patient sleeps.

Hæmoptysis.—Hæmorrhage is a symptom in about 70 per cent. of all cases. It may occur at any period of the disease and is rather more frequent in the young and in males. In the early stages it usually arises from small areas of softening or from erosions of the bronchi. In advanced conditions it comes from erosions of large vessels in the walls of the cavity or from rupture of an aneurysm of a branch of the pulmonary artery. Such hæmorrhages are frequently fatal.

Hæmorrhages occurring in the apparently healthy are obviously the source of anxiety and are frequently difficult to diagnose. There are several types of such cases. First, there are those in which physical examination of the chest is negative and bacteriological examination of the sputum fails to reveal the bacillus. Although in such cases the presence of a tuberculous deposit must always be suspected, many continue in good health for years and have no further manifestations of symptoms as far as the lungs are concerned. Another type comprises those where physical examination is negative after the hæmorrhage, but the sputum, especially a few days after the bleeding, shows the presence of the bacillus. Still another type of cases where the first symptom to attract attention is hæmoptysis is where upon investigation it is found that the health has been failing for some months previous without the patient being aware of the change. These cases show on examination areas of consolidation and bacilli and elastic tissue fibres in the sputum.

Except in the instance of oozing from the erosions of the mucous membrane of the bronchi, the bleeding comes on suddenly, the patient finds his mouth full of blood, and may cough up a mouthful, or a quantity may be ejected in a gush. After the hæmorrhage, small quantities of blood may continue to be coughed up for several days. Large hæmorrhages usually occur only in advanced conditions, and fatal cases are mostly confined to this period of the disease. Fatal hæmorrhages into a large cavity may take place without hæmorrhage from the mouth. The character of the blood is usually frothy, mixed with mucus and of a bright red color, except when the quantity is excessive, when it may be dark.

Gastro-Intestinal Symptoms.—The tongue is frequently furred, but may be clean. The appetite is variable. In incipient cases and sometimes in those moderately well advanced it may be good, but as the disease progresses the digestive powers usually weaken and the appetite fails. Small aphthous patches are not infrequent. A red line on the gums was formerly considered a feature of the disease, but as is now well known it is common to all cachexias.

Extensive ulceration of the larynx, associated with similar condition of the pharynx, may cause dysphagia and seriously interfere with nutrition.

Nausea and vomiting are frequent. They may arise from violent efforts of coughing or from irritation of the pneumogastric nerve, from pressure upon it by caseous glands or stimulation of its peripheral branches, either of pulmonary, pharyngeal or gastric origin.

Diarrhœa is a serious symptom. It may appear early, but generally is more frequent in the more advanced stages. In the first instance it may be the result of indigestion, in the later stages it is commonly associated with tuberculous ulcerations, especially of the lower bowel. Extensive ulceration of the ileum, on the other hand, may exist without diarrhœa. In some cases the diarrhœa is dependent upon the catarrhal inflammation or amyloid degeneration of the mucous membrane.

Emaciation.—In all instances there is loss of bodily weight. This occurs with a varying degree of rapidity. The more rapid the loss the more rapid the process. In the last stage the emaciation is often excessive. With the loss of weight the blood undergoes changes. Anæmia appears early and the count often falls below two millions per cubic millimeter. The blood plates are generally much increased. The leucocytes are also increased. There are, however, no definite or characteristic blood changes.

The Cutaneous Surfaces.—The skin in some cases is dry and harsh. Patches of pityriasis versicolor are quite common, more especially so on the chest and back. Some writers speak of a pigmentary staining, the chloasma phthisicorum, which is more frequently observed when the peritoneum is involved. Clubbing of the finger-nails is often observed in cases running a long course. Œdema of the ankles occurs in the later periods of the disease from nephritic complications, cardiac weakness or phlebitis, generally the former.

Nervous Symptoms.—There are few. The mind usually remains clear until the end. Tuberculosis patients, as is well known, are often inclined to be hopeful. The reverse, however, may be the case, and occasionally relief may be sought in suicide.

Complications.—As every tissue of the body may be attacked by tuberculosis, it follows that the complications may be most numerous, but with the exception of involvement of the larynx and gastro-intestinal tract, comparatively few are of frequent occurrence.

The larynx is very often implicated to a greater or less degree. The tuberculosis process consists of accumulation of cell elements in the mucous and submucous tissues, and may be observed as minute elevations. The first symptom to attract attention is hoarseness; then follows aphonia and dysphagia and other symptoms of the lesion.

Pneumo-thorax is mentioned by several observers as a frequent complication. In the experience of the writer, while not uncommon, it is scarcely frequent. Rupture of the pleura over the thin walls of a cavity is the usual cause of this complication. Its development may be attended with symptoms characteristic of the lesion, or it may come on insidiously without symptoms, as illustrated in Fig. 55.

Enlargement of the bronchial glands, while common, seldom gives rise to symptoms, except in the case of children, where they may cause pressure symptoms.

Acute tuberculous pneumonias are infrequent complications. They may run a hurried course or may be prolonged or atypical.

Bronchiectasis is quite frequent. Gangrene and abscess are occasional terminal complications. Dry pleurisy is very frequent. It may be observed in all periods of the disease. It acts as a conservative process in regard to pneumo-thorax.

Pleurisy with effusion, although much more commonly the precursor of pulmonary tuberculosis, may also occur as a secondary lesion. In some instances it is encapsulated. In rare instances it is hæmorrhagic. Chyleform or milky exudations may also be observed. Purulent effusions are not common, independent of pneumothorax.

Implication of the serous membrane of the heart occur in a small proportion of cases. It is said that endocarditis occurs in about 5 per cent. of all cases.

Secondary implication of the gastro-intestinal tract is such a frequent complication that it has been discussed with the general symptoms. Ulcers of the mouth may be observed, especially on the soft palate, at the base of the tongue and tonsils. Tuberculosis of the pharynx occurs either in association with that of the larynx or independently.

Ulceration of the intestines are frequent, especially in the colon and ileum. In some instances there may be perforation and peritonitis.

Rarer secondary complications are meningitis, peritonitis and amyloid or lardaceous degeneration of the abdominal organs, notably the liver and mucous membrane of the bowels.

In the last stages nephritis usually of a parenchymatous type often makes its appearance.

Mode of Termination.—Chronic pulmonary tuberculosis may terminate in several ways. The usual end is death by asthenia. In many instances the patient passes away quietly, retaining consciousness up to the last. In many again the lungs fill with mucus, becoming œdematous and there is great dyspnœa, preceding unconsciousness and death. Hæmorrhage is probably the next most frequent cause. This fatality arises from erosion of a large vessel or rupture of an aneurysm in a pulmonary cavity.

True asphyxia is scarcely ever observed in the chronic forms of pulmonary tuberculosis, except when pneumo-thorax occurs, but may occasionally be observed in acute miliary and pneumonic tuberculosis.

Syncope is another unusual mode of termination. It may, however, occur in advanced cases upon undue exertion. It may follow pneumothorax from pressure on the heart and thrombosis or embolism of the pulmonary artery.

Cerebral symptoms also terminate a small proportion of cases, namely, hæmorrhagic pachymeningitis, tuberculous meningitis and thrombosis of the cerebral sinuses with meningeal symptoms.

PHYSICAL SIGNS.—*Inspection* may reveal the so-called phthisical chest, which is long, narrow and flat, with the ribs more vertical, the intercostal spaces more narrow than normal, and the cartilages more prominent. In some instances there is a marked depression in the lower portion of the sternum forming a concavity. With the progress of the disease, these features become more pronounced, depressions appear in the supra- and infra-clavicular fossæ with consequent prominence of the clavicles, while, owing to the emaciation, the scapulæ stand out from the body, assuming the appearance sometimes called "winged."

It must not be supposed that the chests of all subjects of pulmonary tuberculosis are of this conformation, for occasionally, as mentioned elsewhere, the disease is observed in persons of good and even fine physique.

The respiratory movement shows restricted motion on the affected side. This may be due not only to consolidation, but to pleuritic adhesions and fibroid induration. There may be also increase in the area of the heart impulse in the second, third and fourth interspace.

As the disease advances, the skin becomes pale, thin, transparent and without subcutaneous fat, the veins appearing with undue prominence.

Palpation shows loss of respiratory movement and increase of tactile fremitus on the affected side, tactile fremitus being increased over tubercular deposits and consolidations.

It should be remembered in comparing the apices that vocal fremitus is normally stronger on the right side than on the left, that it is diminished over marked pleuritic thickening and pleural effusion and increased over cavities.

To obtain the signs of palpation stand behind the patient and place the thumbs in the supra- and the fingers in the infra-clavicular regions then place the hands on each side of the chest. While in both positions, have the patient take a long breath. Thus any loss of expansion can be readily recognized. Again, with the hands in the same position, tell the patient to count or say ninety-nine and thus obtain tactile fremitus.

Percussion gives dulness over areas of consolidation. This change varies with the extent, situation and locality of the diseased area. The points where dulness is first noted are above, over and just below the clavicle, then over the supra-spinous space of the scapula. In these localities, changes in the percussion-note are also more readily observed. Later, the areas of dulness extend downward, involving the middle and the lower lobes.

In the earlier periods of the disease, the percussion-note is higher in pitch. If the consolidated areas are small, with normal lung tissue between, or if there are many small cavities with fibroid induration, there may be slight change in the percussion-note. In recently formed areas of consolidation from cheesy pneumonia the percussion-note has a tubular or tympanitic quality. The same may be observed when the lung has undergone a considerable degree of fibroid change due to excess of connective tissue. Over a cavity of some size it may also be tympanitic. The pitch of the note over a cavity also may undergo change according to whether the mouth is open or closed, constituting Wintrich's sign. Cracked-pot resonance is observed over cavities of large size with thin walls and in those in communication with a large bronchus. Wooden dulness is rare except in cases of long standing with fibroid induration.

In performing direct percussion, especially in thin subjects, a local contraction of the muscles called myoidema may be observed, but it is without special significance.

When percussing the chest, the two sides should always be compared.

Auscultation shows numerous changes of very important significance. In the earliest periods of the disease, the first signs in many instances are feebleness of respiration and cogged-wheel respiration at the apex. Both these features are due to loss of resiliency of the lung tissue, but are in no way distinctive of pulmonary tuberculosis. Prolongation of the expiratory murmur is an important early sign, especially if present at the apex and in association with other symptoms. The pitch is also higher than that of inspiration, which in turn is higher than normal.

In the earlier periods of the disease, a careful comparison with the well side of the chest must always be made.

The next change noted is harshness of the respiratory murmur, and in some cases it is the first recognized. As soon as the consolidated area is sufficiently dense this gives way to bronchial or tubular respiration. At the same time bronchophony appears and with it occasionally pectoriloquy and ægophony. The former if a consolidated area is adjacent to a large bronchus, the latter when in addition to consolidation there is some pleuritic effusion.

With these changes, râles of different varieties develop, the most important and significant being the subcrepitant. In the earlier periods of the disease these are of a decided crackling character. They may be audible only when inspiration is deep, but in pronounced cases are generally always heard and persist to a greater or less degree in some portions of the lung throughout the course of the disease. Sometimes crepitant râles are also heard when there are areas of pulmonic inflammation.

As the disease progresses and the lung softens, loud moist râles may be heard. A very important change at this period is the formation of cavities. The recognition of cavities in the lung is usually not difficult, yet sometimes they are found at post-mortem, where in life their presence was not detected.

The diagnosis of cavities, as far as the physical signs are concerned, depends upon auscultation supplemented by percussion. The latter holds a decidedly secondary position and has already been discussed. The signs elicited by the former may be summarized as follows: Changes in the respiratory murmurs, which are described as cavernous, amphoric and blowing. There are also large, coarse, bubbling râles, gurgles, and on coughing metallic tinkling. Cavernous respiration denotes a cavity of small size with flaccid walls which probably collapse more or less on expiration and expands on inspiration; amphoric respiration, a large cavity with tense walls; bubbling râles and gurgles, fluid in the cavity in sufficient quantity to rise above the opening; metallic tinkling, a large cavity. The latter is described by some as due to the dropping of fluid into a cavity.

Vocal resonance in greatly increased, and pectoriloquy may occasionally be heard, the latter being more decidedly associated with cavities than with consolidations near large bronchi as mentioned.

Pleuritic friction murmurs may be heard at any stage and may occur early. When that portion of the lung which overlaps the heart is involved, a pleuro-pericardial friction murmur may develop, occasionally when this area is consolidated there may be a clicking, râle-like sound synchronous with the heart-beat. This is caused by the heart pressing the air out of the portion of lung in question. The so-called cardio-respiratory murmur may be also frequently heard with pulmonary tuberculosis. It consists of a systolic bruit which is caused by expulsion of the air through the bronchial tubes by the action of the heart. This phenomenon may be best observed during inspiration and in the antero-lateral regions of the chest. In the normal side of the chest, or, if both sides be affected, in the normal portions of the lung, the respiratory murmur is intensified.

Diagnosis.—The early detection of the disease is a matter of the utmost importance, for upon it depends in a large degree the success of treatment. The physical signs of consolidation and crepitant râles, and the symptoms as manifested in persistent cough, expectoration, sweats and loss of weight, are characteristic. It is important to bear in mind that sometimes the physical signs of the disease may be present without the patient's showing any serious impairment of health or being aware of his condition. Positive evidence is the presence of the bacillus in the sputum. When elastic tissue is also found, it is additional evidence, although by no means pathognomonic of pulmonary tuberculosis.

If the result of examination for the bacillus is negative, it does not follow that the case is not tuberculous. That is, while the presence of the bacillus is conclusive, the failure to find it does not necessarily imply absence of the disease. In those instances where there are evidences of consolidation and other symptoms of phthisis, and the bacillus is not discoverable, if the diagnosis of tuberculosis is not positive it should at least be presumptive as far as the surveillance and treatment of the patient is concerned. In all vague and negative cases, the fact that a latent tuberculosis may be creeping on should never be overlooked.

All patients with a hereditary tendency and a phthisical physique in whom there is a persistent cough, or in whom there are evidences of general decline and marked loss of bodily weight, even without other symptoms, should be regarded as liable to tuberculosis.

Hæmoptysis should always be followed by careful and repeated examinations, both physical and bacteriological, that is, after the acute symptoms have passed; and even if such examinations are negative, the patient should be kept under observation for at least six months.

Tuberculin as a test is often unreliable and the results of the latest observations are disappointing. The dangers attending its use must not be overlooked. Latent tuberculosis may assume an active course after its administration.

The spirometer for measuring the capacity of the lungs as a means of diagnosis in the early stages of tuberculosis is uncertain. Potain's sphygmomanoter for measuring the blood-pressure is likewise unreliable, as the blood-pressure in tuberculosis in the early stages varies within a wide range of limitation.

Ehrlich's diazo-reaction, as is well known, may be obtained in some cases of tuberculosis, but cannot be considered as an aid to diagnosis.

In all instances, patients in the early stages of the disease should be made aware of their condition, otherwise the proper treatment cannot be instituted. Their friends likewise should be informed.

The chief sources of error in diagnosis are: malaria, abscess of the lung, bronchiectasis and syphilis of the lung.

Cases of pulmonary tuberculosis with fever, sweats and possibly chilliness, but with feebly defined physical signs, may be mistaken for malaria. Careful examination will remove all possibility of error. Pulmonary tuberculosis, if advanced sufficiently to be attended with hectic, shows evidences of consolidation of the lung, increased rate in respiration and in the majority of instances is attended with its specific micro-organisms in the sputum. Furthermore, examination will not reveal the presence of the plasmodium-malariæ in the blood. Malaria, on the other hand, is not attended with changes in the lungs, examination of the blood will show the plasmodium-malariæ, and there is no change in the rate of respiration. Moreover, the sweats attending tuberculosis belong more distinctively to the night time, which is not the case with those attending malaria.

Abscess of the lung may be distinguished by the following characteristics. It develops in the course of an unresolved pneumonia, or with empyema, or as the result of pyæmia, or traumatism, and is generally situated in the lower lobe on one side only. The sputum, moreover, does not contain tubercle bacilli. Pulmonary tuberculosis, except in the instance of pneumonic tuberculosis, does not immediately follow pneumonia, is more chronic in its manifestations, the early lesions in the majority of instances are in the upper lobe of one lung, later on bóth sides, and the sputum contains the specific bacillus.

Bronchiectasis differs from pulmonary tuberculosis in the greater slowness of its progress. If progressive at all, the absence of the bacilli in the sputum, the evidences of cavities at the base and the presence of cavities before consolidation are conclusive.

Pulmonary syphilis may simulate a tubercular consolidation and thus prove a source of erroneous diagnosis, but this may be avoided by taking into consideration the history of the case, the fact that the gumma is usually situated in the middle and lower lobes and the process is localized, all of which is in contradistinction to tuberculosis in which the lesion is first apparent in the upper lobe and the signs are more diffused. Furthermore, the presence of the bacillus in the sputum removes any element of doubt.

X-RAY DIAGNOSIS.—The X-ray as a means of diagnosis is of great importance. It enables slight changes to be observed in the early stages which otherwise might not be recognized. For the discussion of the subject, including Williams' sign, the reader is referred to Part VII.

Prognosis.—The importance of early diagnosis and its bearing upon the future has already been emphasized. There are, however, many other aspects of the situation which must be taken into consideration. First, the character of the symptoms: A moderate degree of consolidation, slowness and insidiousness of onset, slight fever, little loss of weight, are obviously favorable. Second, absence of heredity, good chest development, previous good health and good digestion are also favorable. As stated in the discussion of treatment, the outcome depends in no small degree upon the power of digestion and assimilation. In proportion as the reverse of these conditions prevails, the prognosis will be correspondingly unfavorable.

Hæmoptysis when recurrent is very unfavorable, as it denotes extension of the ulcerative process and implication of the walls of some artery or arteries. In those cases where it occurs in the latter stages of the disease it may prove to be the immediate cause of death. Under these circumstances, the possibility of its occurrence should always be borne in mind as to treatment as well as prognosis.

A high temperature range denotes a tendency to rapidity of progress, and even in the apparent absence of extensive changes must always be considered as unfavorable.

A temperature range where the remissions fall to subnormal is also unfavorable. A mild temperature with an even range and no subnormal remission is therefore a hopeful indication. When in cases of this type the temperature begins to run at a higher range it signifies that the disease process has become more active. The presence of complications naturally adds to the gravity of the situation, intestinal and meningeal tuberculous involvement being especially unfavorable.

Involvement of the larynx increases the patient's suffering and hastens the downward course of the case. Pneumonia obviously renders the outcome much more grave. When albuminuria develops it is often a precursor of the end. Œdema of the feet and ankles is very unfavorable and is frequently an indication that the patient's forces are nearly exhausted. It may occur without albuminuria.

Environment is of very great importance. The chances are that even in incipient and hopeful cases, unless the patient is placed under favorable hygienic surroundings, the disease will advance to a fatal issue.

The course of chronic ulcerative pulmonary tuberculosis is exceedingly variable. Laennec estimated the average duration to be two years. Pollock gave it at two and a half years. These figures are from hospital practice. In private practice the duration is somewhat longer.

Sometimes in the course of the disease there are intervals in which the fever grows less, the cough and expectoration diminish and there is general improvement.

Spontaneous cure occurs. This is proved by the discovery of old cicatrices, indurative and quiescent healed cavities in persons who have died from other diseases. The cicatrices consist of fibrous tissue. The cavities are found to have undergone shrinkage, becoming surrounded with fibrous tissue. In some instances they are dry; in others they continue to secrete a small amount of purulent matter. In every case of apparent cure, absolute certainty in regard to the complete arrest of the disease can never be assured, as it may break out again at some future period.

Treatment.—PREVENTION.—The greatest advances in the management of tuberculosis have been in prophylaxis, and at the present time the main hope of limitation and eradication of the disease depends upon the strict and widespread application of preventive measures.

The keynote of prophylaxis is struck by bearing in mind that the drying of the sputum,—the freeing the bacillus, allowing its escape in the atmosphere and subsequent inhalation,—is the principal source of infection and the most difficult to manage. It has been stated by eminent authority that the microbe-laden spray which emanates from the consumptive when in the last stages is more virulent than the dried sputum. While this is doubtless true, comparatively few cases of tuberculosis give a history of any such direct exposure; the disease, on the other hand, appearing in the majority of instances without any recognizable source of infection.

There are two aspects to this great question—the public or legislative, and the private or individual. The discussion of the former is scarcely within the province of such a work as this. In regard to the latter, the following measures should be enjoined: The first requirement is the absolute prohibition of expectoration except in receptacles prepared for this purpose. For ambulatory cases, these should consist of tightly-corked, wide-mouth bottles, which the patient carries in his pocket; and, for bedside use, covered sputum cups; in both place a small quantity of a 20-per-cent. solution of carbolic acid, in preference to a solution of bichloride of mercury which hardens the sputum and encapsulates the bacillus.

At least twice in twenty-four hours these vessels should be subjected to heat of over 250° F. of temperature and the sputum rendered sterile. After this they may be cleansed and returned for use. Handkerchiefs for nasal discharges or for general use should be burned, or, better still, "Japanese napkins" may be substituted. Male patients should be smooth shaven lest particles of sputum lodge on the moustache or beard. The patient should always sleep alone in a room without carpets or hangings, and unless in an institution designed for the exclusive care of tuberculosis patients, his table appointments—such as forks, spoons, knives, cups—his clothing and table linen should be sterilized and washed separately.

In all instances the tuberculous patient should reside in an institution, or in some locality where his presence will not constitute a constant menace to others. The non-enforcement of this rule is responsible to a large degree for the spread of the disease.

Children born with tuberculous tendencies should receive especial care as to diet and development. In the instance of a tuberculous mother, nursing should be forbidden and a wet-nurse substituted. In early childhood, milk, eggs and meat-juice should constitute the principal articles of diet. From time to time the child should be weighed and its progress noted. It should spend as much time as possible in the open air, and sleep in an apartment with the windows open all the year round, provided draught can be avoided. A dry, bracing climate, with perhaps a month or two at the seashore in the summer for the air and bathing, usually suits this class of patients. Warm bathing should be practiced daily. After four or five years of age, it should be followed by cold sponging. As soon as the age permits, the child should undergo a system of light gymnastics which tends to develop the chest and inflate the lungs.

The diseases of childhood, especially those which have been complicated with bronchial or pulmonary affections, should receive more than usual attention during convalescence, and the condition of the lungs should be carfully observed until health is fully reestablished. Catarrhal conditions of the naso-pharynx, adenoid growths and enlargement of the tonsils should in all instances receive treatment as the conditions indicate.

The diet of older children with tuberculous tendencies should be most nutritious and generous. The three regular meals should be supplemented in the intervals between the same and at bedtime by milk, some cereal food or eggs beaten up with milk.

GENERAL TREATMENT.—The chief indications are to place the patient under the most favorable hygienic surroundings, to afford him the maximum degree of nutrition which he is able to assimilate, and to institute such therapeutic measures as will influence the morbid process and relieve symptoms.

To fulfill the first condition,—namely, to place the patient under the most favorable hygienic surroundings—implies the selection of a suitable climate and life in the open air.

Climate.—In determining the question of the climate best fitted for a given case, the state of the lungs, general condition, and to some degree the inclination and taste must all be taken into consideration. Not all tuberculous patients should be sent away. Those who are much emaciated and feeble, with extensive consolidation, hectic and subject to night-sweats, should be allowed to remain at home and end their days near friends—to send such away is not only useless, but may add to their distress.

Having decided that the case is suitable to leave home, the next question will be to decide what locality will be likely to produce the most beneficial results. Generally speaking, plenty of pure fresh air of any kind will do good; but an equable climate, with the maximum amount of sunshine, is by all means the best. Dryness is not always essential, as is shown by the good results obtained at Torquay and Falmouth, in England.

The following is a summary of the nature of the climate of different prominent health resorts.

Dry and moderately mild: Adirondacks, Catskills, Alleghany and Cumberland Mountains. Elevation about 1200 feet.

Moderately warm and moderately moist: Western North Carolina, Western South Carolina, Western Georgia.

Moist and warm: Florida, Southern California, Coast region, both equable, Bermuda.

Cool and moderately dry: Minnesota, Dakota.

Cool and dry: Colorado, Wyoming, Montana, Northern New Mexico, Western Kansas. Elevation 4000 to 7000 feet.

Warm and moderately dry: Southern California, Southern Texas.

Warm and dry: Southern New Mexico, Southern Arizona.

Davos, Les Ardents and St. Moritz in Switzerland are high altitude resorts; the Madeira Islands are mild and moist; the Reviera is mild and moderately moist; Egypt and Africa, mild and moderately dry.

Of the American resorts, the Adirondacks are the most popular in this section of the country. The comparative ease of access, the good results attained, the facilities for camp life, and the excellent sanatoria for those who do not wish the latter, are all in its favor. When high altitude is desired, the Colorado resorts are most in favor, but those of Arizona are coming into greater prominence. Resorts of a moderate altitude and moderate degree of mildness find popular and well known representatives in Asheville and Aiken.

In determining the question of climate, the influence of altitude must always be taken into account. The rarification of the air in high altitudes is of benefit in increasing the respiratory movements and dilating the air vesicles of the lungs, thereby increasing the size of the chest; but every case is not benefited by high altitude and a careful discrimination must be exercised.

A consideration that applies to all places is that the soil should be well drained and free from dampness.

The following is a summary of the indications and contra-indications as to high altitude in the climatic treatment of pulmonary tuberculosis:

1. Early and slight apical consolidations, with little constitutional symptoms, are most benefited.

2. More advanced cases with consolidation, but no cavities or any serious disturbances, also do well. When the apices are much involved, the pulse generally over 100 and the temperature about 100° F., a low altitude is better at first. A high altitude may be tried later.

3. Early cases with hæmoptysis, without fever or marked evidences of the disease are benefited, likewise convalescents from pneumonia and pleurisy.

4. Advanced cases should not be sent to high altitudes, that is, those with cavities and pronounced hectic symptoms. A small cavity alone, if apparently not active, is not a contra-indication.

5. Acute cavities, fibroid phthisis, nervous palpitation, emphysema, empyema, albuminuria and diabetes are contra-indications. A valvular murmur with no sign of enlargement is not a contra-indication. Nervous persons with insomnia are better on a low level. Laryngeal complications are generally considered as contra-indications, but such often do as well in high altitudes as elsewhere.

While the above may serve as a general line of indications, it may be said in conclusion that when the patient has a good family history, and the disease is limited to the apex, the chances are favorable whatever may be the climate, if the patient lives out of doors.

If the lungs are much involved and cavities have formed, the chances of permanent cure are small. Such cases generally do better in mild climates.

Open-Air Treatment.—The value of life in the open air in the treatment of pulmonary tuberculosis is now so well recognized that it may be said to be the first feature for consideration. It may be carried on by change of residence to some suitable climate, by camping, at a sanatorium, or at home.

When a patient has taken up his residence in a good locality for the sake of the climate, and is not in camp or at a sanatorium, he should place himself under the care of some local physician and live according to rule. He should in all instances live out of doors continuously.

1. Camp Life.—The most effective method for carrying out open-air treatment is that of camping. It promises the best prospects for restoration, for it affords the fullest measure of out-door living. The novelty of the life and the light pleasurable occupations that go with it also help in no small degree. It is, however, of limited practicability, as many are unable to adopt such a plan of living.

The class of cases which is suitable for camp life is composed of those in the incipient stage, and the earlier periods of the second stage. The first are generally permanently restored, while in many of the second the progress of the disease is arrested for a considerable period.

Camping may be conducted on two plans. First, select a favorable site and there establish the camp for the season; second, what might be called "gypsy camping,"—that is, with a wagon outfit and tent, travel from place to place, and establish the camp every few days in a different locality. The first plan may be undertaken with only two persons; the second, with not less than three. The migratory plan necessitates a good horse and a good covered wagon, in addition to the outfit of tents, blankets, waterproofs, hammocks, and the usual camp appointments and accessories. The other plan requires the same outfit, except the horse and wagon.

The best season of the year for camping is from spring to autumn. Having decided upon the site, carefully locate the camp on dry soil, avoiding exposure to high winds and bad drainage.

When winter approaches the patient must not return to his home in the city, but should continue open-air treatment in some good sanatorium, and to complete the cure should camp out for a second season. A better plan when winter approaches is to go South and camp in the pine regions of North Carolina or Georgia.

2. At Home.—There are many patients, however, who cannot afford to leave home. How then can the open-air treatment be carried on? If in the country, or in a country town, that is not a difficult matter. If there is a porch with a southern exposure, have it enclosed for the winter with glass which can be opened or shut at pleasure. When the weather is rainy or windy, the side exposed to the wind or rain should be closed and the other opened wholly or in part. In summer the glass can be removed. On the porch or veranda the patient well wrapped in rugs and blankets should pass the entire day. At night he should sleep in a room with the window open, more or less, according to the weather, but always open some. In mild weather he can sleep in the porch. When the enclosed porch is not possible or practical, a so-called sun-trap may be constructed at slight expense. This consists of a structure of light lumber about six feet square, built like a lean-to shed, with one side open and the three remaining sides enclosed, with a window in each, protected by sliding sashes and curtains. This arrangement can be moved in a yard in such positions as to avoid the wind and receive the sunlight. In a closely built city, a room with a southern exposure, with the window kept open, is the nearest approach to open-air treatment which can be had.

3. Sanatoria.—These institutions have been so successful abroad and in certain localities in this country that they are now generally

recognized as a most important advance in the treatment of tuberculosis. The patient has the great advantage of having his daily life and diet carefully regulated and of being under constant medical supervision. At all these institutions the open-air treatment is rigorously carried out, the buildings being constructed with this end in view.

Diet.—Nutrition must be considered of equal importance with open air. In fact, the outlook depends in no small degree upon the digestion. If gastric symptoms are prominent, and there is more or less anorexia, the progress of the case is obviously much impeded. A change of air or a sea voyage may restore the appetite. Frequently camp life stimulates the appetite to a marked degree. When the digestion is good an excellent general rule is to give three moderately solid meals a day, with some liquid or semi-liquid nourishment at about 10 A.M., 2.30 P.M. and on retiring, that is, about an hour to an hour and a half after regular meals—thus avoiding destroying the appetite for the next meal.

When the digestion is feeble, as is often the case, especially when there is fever, a liquid or semi-liquid diet usually proves most acceptable. The best liquid food is milk—koumyss or matzoon may be substituted. When milk does not agree with the patient, lime water may be added. Some prefer peptonized milk.

While a large amount of nutriment is indicated in all these cases, caution is necessary on account of the delicate condition of the stomach, lest the digestion be still further disturbed. When vomiting is frequent and there are evidences of gastric irritation, the stomach may be washed out and a quantity of pre-digested food administered through the tube, according to Debove's method. Debove, after washing out the stomach with cold water, administers one litre of milk, one egg and one hundred grammes of finely powdered meat. This is given three times a day. The results are often decidedly beneficial.

When the digestion is strong, the patient may take ordinary diet, selecting the most nutritious articles. Red meats, especially underdone roast beefsteak, mutton and venison, should be preferred. He should eat the fat along with the meat. Poultry, oysters and fish may be taken for a change. The patient should use butter freely. Milk should in all cases be taken in liberal quantities, in addition to the regular diet. It is a good plan, on wakening in the morning, after washing the mouth with some antiseptic fluid, to take a glass of warm milk. Milk should be taken also at bedtime and on wakening during the night, and at various times during the day *ad libitum*. It may be varied by mixing with some cereal food.

Eggs should form an important item. They may be taken in all forms except fried. Raw eggs have a good effect in restoring nutrition, and if possible should be used in liberal quantities. An exclusive diet of two dozen eggs daily has been recommended, but there are few who could tolerate it.

Vegetables, except cabbage, fruits, especially grapes, simple puddings containing milk should also constitute an important part of the diet.

While the above constitutes the general line of diet, it is obvious it must be more or less modified according to the requirements of each individual case, especially if there is a tendency to diarrhœa.

Alcoholic stimulants are often useful, but should be prescribed with discretion. Patients with incipient disease are better without them. Moreover, the danger of forming the habit should always be taken into consideration. A little red wine, however, may be allowed with the dinner and is often helpful. Advanced cases are generally benefited by small quantities of alcohol. Eggnog and malt liquors are usually decidedly beneficial. Of the latter, porter is especially to be commended, but, if objectionable, some of the preparations of malt extract may be substituted.

Articles to be avoided are pastries, rich puddings, heavy sweets, cake, fried meats, corn beef and fresh pork.

The Clothing.—The clothing of a phthisical person should receive especial attention. The underwear should be of wool—heavy or light, according to the season of the year. Heavy chest protectors are not recommended. Great care should be exercised in regard to keeping the feet warm and dry. In winter, when out-doors, a bountiful supply of wraps and rugs should be on hand. In rainy weather waterproofs and rubber overshoes should be worn.

Warm or tepid water should be used for bathing, which should be practiced daily in the form of sponging, especially if there are night-sweats. In addition, the chest should be sponged with cold water and rubbed briskly.

Rest.—Rest also forms an important element in the treatment of tuberculosis. If the patient is easily fatigued, or if exercise is attended with rise of temperature, it is advisable that he should be put to bed for at least a portion of the day; or, better, should spend the entire time in a steamer chair out of doors in the sunshine. This should be kept up until the temperature falls below 100° F. and does not rise above same. Sometimes six to eight weeks, or more, will be required to attain this result. Such patients should on leaving the bed do so very gradually, sitting up for short intervals at a time, increasing gradually day by day. They should also be very careful about beginning exercise, and should do so little by little.

Occupation and Exercise.—In the incipient and early stages of the disease the patient is greatly assisted by some light out-door pursuit which occupies both body and mind. One of the advantages of camp life is that, with its little daily duties, fishing, hunting, boating, etc., it fulfills this condition so amply. For those not camping out, light gardening, driving, riding, etc., may be suggested.

Walking should be allowed according to the patient's strength. The rule in regard to walking and all forms of exercise is that it should not be sufficient to cause fatigue, and that if the temperature afterwards rises to 100½° F. it is an indication that the walk or exercise should be limited. Exercise which tends to develop the chest should be practiced daily, and should form an important part of treatment in early cases.

THERAPEUTICS.—The absence of any remedy or line of medication which can be regarded as specific in the treatment of pulmonary tuberculosis is too well recognized by the profession to require comment. This, however, does not signify that therapeutic agents should be discarded, for there is no doubt that while alone they fail, in conjunction with open-air treatment and superalimentation they act as valuable adjuvants. In early incipient cases, with few and ill defined symptoms, fresh air and alimentation alone are often sufficient.

Treatment according to the principle of *Similia*, while obviously not anti-tubercular, seems none the less, when drugs which distinctly act upon the pulmonary parenchyma are employed, to meet the morbid conditions in many ways. The following are some of the principle remedies of this class :

Arsenici iodidum is often of signal service. It acts best in the earlier period of the disease when there is rapid loss of weight and marked prostration, together with hectic and diarrhœa. The remedy acts after the formation of a cavity as well as when one is threatened.

Arsenicum album, according to Allen, is a valuable remedy for the cachexia which leads to chronic disease of the lungs. It seems, however, when the disease is established, better suited to somewhat advanced conditions, characterized by the presence of a lymphatic nervous temperament, great debility, emaciation which comes on rapidly, restlessness, mental anguish, fear of death, waxy pallid countenance, craving for acids, great thirst, drinking frequently, but in small quantities, offensive wasting diarrhœa, burning pains, nausea, vomiting and cough, usually dry with a sense of irritation like the inhalation of sulphurous gas.

Iodidum is indicated when there is enlargement of the glands, dry cough, hoarseness, heavy night-sweats, pain and stitches in the larynx, ravenous appetite, notwithstanding which there is continuous loss of flesh.

Antimonii iodidum is an important remedy and frequently acts with marked benefit in cases where there is profuse expectoration, with the constitutional symptoms indicating iodine. Tendencies to consolidation, with profuse muco-purulent expectoration, are not infrequently relieved by it.

Stanni iodidum, after the iodide of arsenic, and the iodide of antimony, is a most valuable remedy. Its special indication is profuse expectoration containing abundant pus cells, greenish or yellowish in color, sweetish in taste and easily expectorated, sometimes coming up in balls. Stannum may be administered on the same general indications. Both remedies are used in hectic and emaciation. Sense of weakness in the chest, fatigue on talking are additional but minor indications.

Lime ; of all preparations of lime the hypophosphite stands first. It may be administered with good effect in the early stages of pulmonary tuberculosis in young subjects with delicate tissues and scrofulous tendencies, when the condition is characterized by a slight rise of temperature, moderate degrees of cough, flushed cheeks and tendency to sweating. Three or four grains of the first decimal trituration may be used several times a day. Calcium carbonicum, in the third or the sixth decimal trituration, is indicated by the presence of free perspiration, rapid emaciation, loose rattling cough, soreness of the chest, painful on percussion, hoarseness and diarrhœa. It should be employed when these symptoms are present in the early stage of the disease, in subjects with loose, flabby tissues, acid dyspepsia and cachectic tendencies.

Phosphorus is helpful in miliary tuberculosis, attended with afternoon fever, flushed face, dry short cough, hoarseness, debility, rapid breathing, sweats and progressive emaciation. The presence of soreness in the larynx and trachea, diarrhœa, especially after meals, palpitation, blood-streaked sputum, are additional important indications. The remedy is said to act better in tall, thin persons, and in the young.

Silicia may be used in the suppurative stages with offensive, profuse expectoration, cavities, fever and profuse sweats.

Acidum sulphuricum; in the last stages when there is aphthous ulceration of the mucous membranes, with great debility, sweats and venous hæmorrhages from the orifices of the body, this remedy will often palliate. It may be used in the second or third decimal dilution. It may also be applied to the ulcerating surface.

Kali carbonicum; the presence of sharp stitching pains in the chest, with dry cough, or cough with scanty expectoration, hoarse in the early morning, cough with tickling in the throat, cutting pains in the chest after lying down at night, extending to the left hypochondrium, are the chief symptoms pointing to this remedy.

Other remedies which may be prescribed according to their special indications are sulphur, sanguinaria, antimonium tartaratum, pulsatilla, lycopodium and kali iodidum.

Certain other drugs are regarded as especially beneficial, and in some instances are no doubt effective. The most important of these are as follows:

Creasotum is one of the most popular and widely used remedies. It does not appear to have any decided effect upon the tuberculous process, but it is of benefit in stimulating nutrition. It may be administered in capsules, beginning with one minim doses three times a day, increasing gradually to eight or ten minims. It may also be given in solution with alcohol and cardamon, or in the form of the carbonate in fifteen-grain doses.

Guaiacol may be given as a substitute, but does not appear to influence the progress of the disease, although it lessens the cough and expectoration.

Iodoform certainly possesses a decided antagonism to the tubercule bacilli, and its use in pulmonary tuberculosis has not been without success. The writer has used it in a number of instances in the form of inunction, according to the following formula:

℞ Iodoformi,		℥ss.
Ol. anisi,		♏x–xxx.
Ol. gaultheriæ,		ʒij.
Ol. olivæ,		
Lanolini,	āā	℥v.

Administer in the same manner as mercurial inunction, rubbing the flexor surface of one arm one day, that of the other arm the next; that of the thigh the next, and so on. About one drachm should be used at a time. The process should be kept up for weeks. No injurious effects have been observed. Europhen or iodol may be substituted for iodoform in private practice.

Ichthyol at one time was much in vogue. The writer has used it quite extensively, commencing with three drops of the crude drug in capsules three times daily, and increasing it up to the point of intolerance, which was generally reached when eighteen or twenty minims were given. The result of observation in some forty cases showed that the remedy, when the dose was not large enough to cause gastric disturbance, was in some instances decidedly beneficial, while in others it was without effect. Its administration must be entirely empirical, as so far no especial indications were discovered which would seem to individualize its use.

Strychnia is occasionally useful in doses of one or two grains of the second decimal trituration three times daily. It appears to act beneficially in cases characterized by weak digestion and nervous symptoms.

Cod liver oil has met with disfavor in some quarters, especially in certain sanatoria. As a remedy against pulmonary tuberculosis it is of no direct value, but it certainly helps in improving the nutrition. It is recommended especially in public institutions, where the dietary is necessarily somewhat limited. Fever and gastro-intestinal disturbances are contra-indications to its use. When it disagrees, cream may be substituted.

TREATMENT OF SPECIAL SYMPTOMS.

Fever.—Patients with temperature over 100½° F. should be kept in bed. When the weather and circumstances permit the patient should be placed in a cot out of doors.

When the temperature is high and it is considered advisable to treat this symptom, the tincture of baptisia, or arsenite of quinine, may be used with good effect. The latter may be administered in one-grain doses of the second decimal trituration every two hours. Sponging with tepid water and alcohol may be used when more immediate effect is desired. The coal tar derivations, phenacetine, phenalgen, or acetanilid, in two- or three-grain doses, will reduce the temperature for the time, and may be used occasionally, but are not recommended for regular or general administration.

Cough.—Cough is often a very troublesome symptom and requires special attention. Much relief can be obtained by observing the following simple general rules :

Avoid excitement, talking or any exercise before bedtime. Before retiring, let the bed be warmed and the room well ventilated. A woolen nightgown should be worn instead of one of cotton. A glass of hot milk should be taken at bedtime and during the night on waking. A small amount of stimulant may be used in many instances with good effect. The patient should be taught how to control the cough, namely, to avoid coughing when there is no mass of accumulated secretion in the lung, and to encourage it when there is. The morning cough should therefore be encouraged. It may be greatly assisted by the glass of warm milk. When cough is excited by taking food, sometimes causing vomiting, the patient should rest in the recumbent position for an hour before and after meals. He may also take his food while reclining.

When it is necessary to give drugs to relieve cough, on account of the loss of sleep which it occasions at night, which is frequently the case in the last stages, hyoscyamus in the second decimal, or codein, $\frac{1}{4}$ grain, or heroin in $\frac{1}{20}$- to $\frac{1}{8}$-grain doses, should be considered. Other useful palliatives for the last stages are a mixture of chloroform in whiskey, and morphine in $\frac{1}{8}$- to $\frac{1}{6}$-grain doses. Remedies of the sedative class, while often necessary in the later stages as palliatives, should always be given cautiously, for the accumulation of secretion in cavities must be relieved.

Inhalations are often useful, particularly when the secretion is profuse. Sometimes an anodyne gargle relieves for a time, especially if the cough appears to be aggravated by ulceration of the pharynx. For this purpose the following is suggested:

℞ Tr. opii camphoratæ,
Aq. menthæ piperitæ,
Glycerini, ... āā ℥j.
Sig.—Dilute with water and gargle.

Pain in the Chest.—Bryonia is the chief remedy. Kali carb., aconite, sulphur, actea racemosa should also be considered, according to the prevailing individual indications. Strapping with adhesive plaster, or painting the chest with the tincture of iodine and the application of galvanism are useful measures. For the chest pain in advanced cases, soothing liniments and embrocations may prove helpful. When the pain is distinctly of a myalgic character, phenacetine, phenalgen, in two-grain doses, or the so-called migraine tablet may be used with good effect.

Sweats.—As the presence of sweats tends greatly to reduce the patient this important symptom, when excessive, demands particular attention. While not infrequently the remedy indicated, according to the totality of the symptoms and pathological condition, will control this condition, special treatment will also often be required. For this purpose the following remedies are recommended: Agaracine, first decimal, one or two grains; dilute phosphoric acid, fifteen drops in half a glass of water; pilocarpine, second decimal, one grain; atropin, one hundred and fiftieth or two hundredth of a grain hypodermically; camphoric acid, thirty grains; picrotoxin, the second decimal, half a grain; aromatic sulphuric acid, fifteen drops in cinnamon water. These remedies are best given about three hours before the expected sweat.

Gastro-Intestinal Disorders.—When the digestion is feeble the diet must be changed as previously suggested. A semi-liquid diet with pre-digested foods will be indicated. Koumyss, milk and lime water, peptonized milk, eggs, toast and meat broths should constitute the principal articles. For flatulence, vomiting and pain after eating, nux vomica, carbo vegetabilis, bismuth and pepsin, papoid, colocynth, mercurius and ipecacuanha are useful.

For diarrhœa, arsenicum is one of the chief and most effective remedies, in the second to the sixth decimal trituration; sometimes, in the form of Fowler's solution, in one-drop doses, it will act better. Cuphea in tincture, and arsenite of copper in the second decimal trituration, for diarrhœa with nausea, vomiting and cramps. When the evacuations show the presence of undigested food, in addition to arsenic, cinchona, ferrum phos. and ferrum ars. may be considered. Other remedies are burnt brandy, salol, bismuth and opium, but the latter should generally be avoided.

When the evacuations are very offensive, a dose of castor oil may be administered, with the view of removing irritating matter, after which the selected remedy may be given. An excellent measure when other remedies fail is intestinal irrigation with normal saline solution or boracic acid.

Hæmoptysis.—The treatment discussed under the general subject of Hæmoptysis is here indicated. The reader, therefore, is referred to that article.

Dyspnœa.—This symptom seldom if ever requires special attention, except in the last stages, when diffusible stimulants, especially ammonia and morphine hypodermically, afford relief.

Stomatitis.—This complication is sometimes encountered in the later stages, and is best met by antiseptic mouth washes containing eucalyptus, myrrh, boracic acid or potassium chlorate.

Dysphagia.—In the last stages, when there are severe laryngeal complications, this condition may require special palliative treatment, viz.:

℞ Morphiæ, hydrochloratis,		
Sac. lactis,	āā	gr. xv.
Gum. arabici,		gr. xv.

Sig.—Use half a grain before meals by insufflation.

℞ Mentholi,		ʒijss.
Ol. amygdalæ dulcis,		
Vitelli ovi, (about twelve yolks),	āā	℥j.
Orthoformis,		ʒiij.
Aq. destillatæ,	q. s. ad.	℥iij.

Sig.—Cleanse part and insufflate.

Lactic acid, in strengths varying from 10 to 100 per cent., beginning with a weak solution, is one of the most effective local applications. Swallowing small pieces of ice will sometimes allay the spasmodic form.

LOCAL TREATMENT OF THE RESPIRATORY TRACT.—In advanced cases when the expectoration is profuse and offensive, and cough troublesome, relief may be obtained by inhalation of some antiseptic. The selected inhalant, properly prepared and placed in an inhaler, should be inhaled without effort for an hour. The best remedy for this purpose is beechwood creasote. It should not, however, be used when there is a tendency to hæmorrhage. The following preparations are recommended:

Creasote, chloroform and alcohol, equal parts; or
Creasote, ethyl iodide and terebene, equal parts; or
Creasote, eucalyptus oil and terebene, equal parts.

Ten to twenty drops of either of these formulæ may be placed in an inhaler. Guaiacol may be used in like manner. The following is also recommended:

℞	Mentholi,	
	Thymoli,	
	Acid. carbolici,	āā gr. v.
	Ol. eucalypti,	℥ij.
	Ol. pini silvestri,	℥viij.

Sig.—A teaspoonful to a pint of boiling water and the vapor inhaled, or twenty to thirty drops inhaled from cotton.

Other useful inhalants are tar water heated and the vapor inhaled; compound tincture of benzoin, a drachm to a pint of boiling water. In most instances inhalation will lessen the cough and the amount of secretion, and thus act as a useful palliative.

3. Fibroid Pulmonary Tuberculosis.

Fibroid pulmonary tuberculosis is that variety of the disease which is characterized by hyperplasia and contraction of the connective tissue of the lungs and slowness of the morbid processes. It bears an intimate relationship to pulmonary fibrosis, but must be distinguished from it, that is, fibrosis of the lung may be either tuberculous or non-tuberculous.

Ætiology.—The disease originates in two ways. First, as a fibro-tuberculosis, in which, after infection of a previously healthy lung, the degenerative processes are of a primarily fibroid nature. Second, as a tuberculo-fibrosis, where the disease follows chronic ulcerative tuberculosis or tuberculous pleurisy.

The disease is more frequently observed in those of fair physique who do not evince heredity, and who appear to have a certain amount of resistive force against the bacillus, notwithstanding its power to cause infection. It is more frequent among men than women and in those whose occupations require them to inhale quantities of dust.

Morbid Anatomy.—The tubercles form in the same manner as in the other varieties of pulmonary tuberculosis, but instead of undergoing caseation and softening the tubercular masses take on a fibroid change, becoming transformed into firm, hard, deeply pigmented bodies. These bodies are found in the walls of the bronchi and the peri-bronchial tissue. They frequently coalesce and new masses form on their surface, resulting in a racemose arrangement ("Carswell's Grapes").

The interlobular connective tissue undergoes characteristic changes. It first proliferates, then contracts and indurates. Bands of tough, grayish fibrous tissue form in the lung structures. The bronchial mucous membrane thickens, the lumen of the tubes and alveoli is encroached upon in some places and dilated in others, and sometimes calcareous deposits and ulcerating cavities occur. Cavity formation, however, is not common unless the process has begun as an ulcerative type. When present, the cavity is usually surrounded with deposits of fibrous tissue which cause more or less shrinkage of its dimensions. These changes lead to both bronchiectasis and emphysema, patches of the latter being commonly present.

The seat of the process is usually at one apex, from whence it extends downward, involving the lung to a greater or less degree. The involved portion becomes markedly shrunken, sometimes to one-third its natural size.

The pleura is thickened and adherent. The heart is exposed by the retraction of the lung tissue, and frequently dilated, either the right side alone or the whole organ. The liver and spleen are often displaced upward.

The apex of the other lung is usually also somewhat diseased.

Symptoms.—The disease manifests itself very insidiously. Cough usually appears early, but it is not severe, being more of a hacking. It lacks the paroxysmal character of that of the ulcerative variety. Although the cough increases as the disease progresses, it seldom becomes excessively troublesome. Frequently, even in cases of many years standing, after having in the morning cleared the bronchial tubes of the accumulated secretion of the past night, the patient coughs but little during the remainder of the day. There is always more or less chronic bronchitis present, which necessarily modifies the cough and expectoration. Occasionally there is hæmorrhage.

Expectoration may be absent for a considerable time, but ultimately makes its appearance; or it may make its appearance early. At first it is mucous and muco-purulent, later it becomes decidedly purulent. Sometimes it is streaked with blood. The quantity is not usually excessive. When there is bronchiectasis it is more abundant and often offensive. Microscopical examination does not reveal the bacilli in large quantities and not infrequently several examinations will be necessary before any are found.

The general nutrition in the early period of the disease does not usually suffer in a marked degree, but ultimately the patient becomes thin and anæmic.

The respiratory rate is somewhat accelerated and there is shortness of breath on exertion, but true dyspnœa is absent unless there is an excessive degree of emphysema.

The pulse shows little increase at first, but later it becomes more rapid, with the changes incident to dilatation of the heart. The right side of the heart and sometimes the whole organ may become hypertrophied and dilated. Murmurs may be present, and subsequently insufficiency may develop.

Fever is absent or very slight. In advanced cases it may arise from absorption of the purulent secretions. Sweats may be present from the same cause, but they are never so prominent as in the ulcerative variety.

Diarrhœa is absent. The course of the disease is exceedingly slow, varying from four or five to ten, twenty and even thirty years. Notwithstanding this extreme chronicity, the tendency is downward, and the patient may die from exhaustion and sepsis from absorption of the purulent secretion, from cardiac complication, or amyloid degeneration of the kidneys.

PHYSICAL SIGNS.—*Inspection* shows retraction of the supra- and infra-clavicular spaces and more or less of the whole chest. The shoulder of the affected side may be lower than that of the other. The heart is usually drawn toward the affected lung. If the left lung is the seat of the process, owing to the retraction, it exposes the heart and in consequence there may be marked pulsation in the second, third and fourth interspaces.

Palpitation shows increase of vocal fremitus over the affected area. *Percussion* gives the signs of dulness common to pulmonary consolidation. Frequently, however, the percussion-note may display a sort of flat tympany, due either to the presence of a large sacculated bronchus or areas of emphysema or to excessive contraction of the lung.

Auscultation gives feebleness of the respiratory murmur in some areas, bronchial breathing in others and fine crackling crepitant râles in still others. The signs of cavities, which are usually small and dry, may also exist.

Diagnosis.—The diagnosis at first may be difficult, but later it is easy. The physical signs as observed on auscultation, the comparative mildness of the symptoms, the extreme slowness in the course of the disease and the presence of the bacillus are conclusive.

The main point of distinction is to determine whether the disease is a tuberculous pulmonary fibrosis or fibroid pneumonia. In many instances this is difficult, for clinically the two diseases are almost identical. The point of difference is the presence of the bacillus in the sputum. Inasmuch as the examination of the sputum in tubercular pulmonary fibrosis may frequently fail to reveal the bacillus, while subsequently examination may show its presence, it is very important in all negative cases that repeated examinations should be made. Additional points of differentiation between the two forms of pulmonary fibrosis are discussed under the head of Fibrosis of the Lungs.

Syphilis of the lung may simulate tubercular fibrosis, but the history of the case and the presence of accompanying syphilitic lesions usually prevent error.

Prognosis.—Under favorable conditions, early in its course, the disease may be arrested. Complete recovery, however, seldom occurs. There may be long periods when the progress appears to remain quiescent, then it lights up again. As far as life is concerned, patients with pulmonary fibroid tuberculosis may live for many years, although it is obvious that the general health becomes impaired, and that there is always a tendency to the development of acute pulmonary affections and cardiac disease. In old cases the danger of amyloid degeneration, or sepsis from absorption of the accumulated secretions, must always be taken into consideration. Some observers mention danger from fatal hæmorrhage, but that is very infrequent.

Treatment.—The same general lines of treatment as discussed under the ulcerative variety are applicable to this form of the disease.

FIBROSIS OF THE LUNGS.

SYNONYMS.—*Fibroid Pneumonia, Chronic Interstitial Pneumonia, Cirrhosis of the Lungs, Fibroid Induration of the Lungs.*

Fibrosis of the lungs is a chronic process characterized pathologically by proliferation of the connective tissue and clinically by evidences of impairment of the function of the lungs.

Ætiology.—The disease results from continued irritation of the lung tissues from any cause, and originates as a primary or as a secondary process. Primary pulmonary fibrosis arising from the inhalation of irritating particles of dust is discussed under the head of pneumonokoniosis. The secondary variety is that generally implied when the term fibrosis of the lungs or any of its synonyms is employed and is the subject of the article. It arises in connection with many forms of chronic pulmonary diseases, as the sequela of croupous and catarrhal pneumonia, from extension of chronic disease of the pleura and from syphilis.

The fibroid changes may involve a limited area of the lung, or one entire lung or portions of both, hence the disease is accordingly classified as local or diffuse.

Local pulmonary fibrosis is common. It may be found to a greater or less extent in all chronic diseases of the lungs. It is constant in chronic pulmonary tuberculosis where it is a conservative process. In cases where the tuberculosis process has been arrested, the involved areas are found to have undergone fibroid change. In fatal cases more or less fibrosis is also found, showing the futile effort of nature to stop the course of the disease. Localized areas of fibroid change are also found in association with emphysema, bronchiectasis, abscesses, tumors, gummata and hydatids.

Diffuse fibrosis of the lungs is observed after catarrhal pneumonia, where it may follow either form, especially in children, constituting what is sometimes called chronic broncho- or catarrhal pneumonia. It may also appear as a sequela of croupous pneumonia, although it is generally admitted that this is rare. Chronic congestion of the lungs, such as sometimes occurs in valvular diseases of the heart, particularly that of the mitral, not infrequently leads to fibroid changes in the pulmonary tissues. Chronic pleurisy may sometimes be followed by similar changes constituting what is known as pleurogenous fibroid pneumonia. Syphilis of the lung presents the features of fibroid degeneration, but is considered separately.

Neoplasms and aneurysm by compression may cause either local or diffuse pulmonary fibrosis.

Morbid Anatomy.—The origin of the pathological process is as follows : The primary inflammation is followed by hyperæmia of the connective tissues with small cell infiltration and the formation of fusiform cells ; these subsequently change or develop into connective or fibrous tissue. The tissues contract later and its blood-vessels become obliterated until finally there results a dense fibrous mass. In consequence the air-cells in the involved areas are compressed or more or less entirely obliterated. The contraction of the tissues is frequently followed by dilatation of the bronchi (bronchiectasis). Pulmonary aneurysms are not infrequent in these cavities. Cases are observed in which the lung is changed to a mass of bronchiectatic cavities and the fibroid process is confined for the most part to the root.

In the local forms the changes are lobar, or broncho-pneumonic, and the fibroid areas are scattered throughout the lung. In the diffuse variety, one lobe, or the greater portion of one lobe, may be affected. The process is usually unilateral when lobar, but when more extensive it may affect both lungs, one much more than the other. Sometimes the lung is much shrunken and lies lightly against the spine. In this condition the heart will occupy the affected side, being drawn out of place during the course of the disease. There is usually considerable hypertrophy, especially on the right side. The pulmonary artery may show evidences of arterio-sclerosis. The lung on the other side may be over-distended and emphysematous.

The pleura is generally involved showing the presence of adhesions which are sometimes very tough and sometimes only moderately so; pleuro-pericardial adhesion may also be present.

On section the affected areas cut with difficulty and disclose hard, dry, airless, somewhat shiny surfaces, which are usually light gray in color, but sometimes reddish.

Microscopical examination of an affected portion shows thickening of the alveolar walls, and compression and obliteration of the air-cells. The latter when not entirely destroyed are seen to contain epithelial cells, granular matter and fat granules. The peri-bronchial tissues share in the fibrous process.

The mucosa in cases of long standing may undergo ulceration in patches, especially in the dilated portions. This is due to retention of bronchial secretions. In advanced cases occasionally gangrene results.

Amyloid changes of the viscera are found in some instances.

A recticular form is mentioned by some writers. In this variety fibrous bands are found scattered symmetrically throughout both lungs. It is very rare.

Symptoms.—The early manifestation of symptoms vary according to the primary lesion and the extent of the lung involved. In slight conditions of fibrosis, such as occur in arrested incipient tubercular infections, there are usually no symptoms. When there is a considerable degree of the lung involved, one of the earliest symptoms is that of cough, which increases as the disease advances. At first it is dry, but later there is a muco-purulent expectoration which may be quite copious in the morning. Sometimes but rarely it is blood streaked. Shortness of breath is also an early symptom. It arises on the slightest exertion, but when the patient is resting it generally ceases. The rate of respiration, however, even when quiet is more or less accelerated. The general health shows a decline,

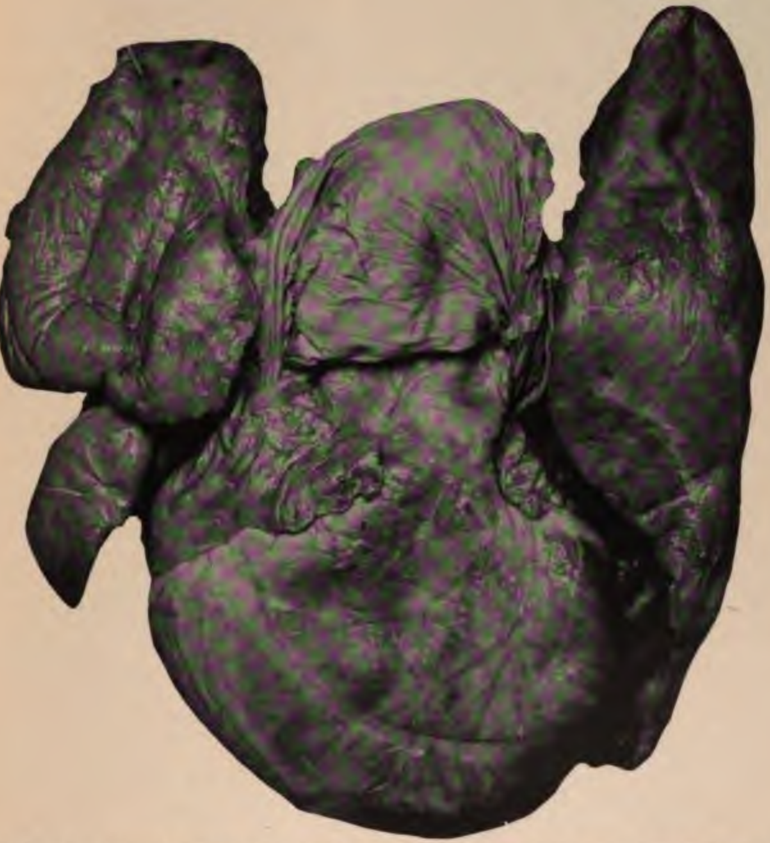

FIG. 51.—Fibroid degeneration of the lower lobes of the right lung, showing atrophy of the same. The illustration also shows marked dilatation of the heart which was the cause of death. The pericardium is lifted showing the heart.

the patient loses flesh and strength. Fever is absent. The pulse at first is slow, but later is rapid. In rare instances there are hæmorrhages. All these symptoms are of slow development and in spite of their presence the patient may be able to enjoy life to a considerable extent and live for many years. The general health, however, will be much below the normal standard, and there is a decided susceptibility to changes of the weather and a tendency to attacks of acute bronchitis. In cases of long standing bronchiectasis often develops. When this occurs there may be fever due to retention of the bronchial secretions and paroxysms of cough, such as usually attend this condition. The expectoration now may become fetid.

Clubbing of the finger-nails may occur. The course of the disease may extend over many years. It tends more or less to an ultimate fatal termination. If no intercurrent disease destroys life, the obstruction to the circulation of the lungs gradually causes dilatation of the heart, which in turn may be followed by the symptoms common to failing compensation.

In cases of long standing general pulmonary fibrosis, when the patient is well along in years and the weakness of age is added to that induced by the disease, retention of the secretion in the bronchiectatic cavities may give rise to a condition of sepsis and the patient may die of auto-infection and asthenia.

PHYSICAL SIGNS.—*Inspection* shows more or less general emaciation and changes in the affected side, namely, retraction with approximation of the ribs, loss of motion on breathing and drooping of the shoulder. The healthy side is enlarged, the spinal column bowed and the heart displaced toward the diseased side, being drawn down by the shrinkage of the lung. When this occurs the impulse may be visible in the second, third and fourth interspaces.

Palpation gives increase of vocal fremitus except when the bronchi are obliterated or when there is excessive thickening of the pleura.

Percussion is variable. Sometimes the note is decidedly flat, especially at the apex, sometimes it is flat and high pitched or flatly tympanitic. These changes are found in various cases and are due to marked shrinkage and thickening of the lung. Over a bronchiectatic cavity there may be amphoric resonance. On the opposite side there is usually hyper-resonance.

Auscultation gives a variety of signs which vary according to the extent of the disease. At the apex the respiratory murmur is tubular. Towards the base it becomes less strong, while at the base itself it is feeble. Râles, sibilant, bubbling and sub-crepitant, are audible in various areas, more especially in the middle and lower portions of the lungs. Of these signs, bronchial or tubular breathing and sub-crepitant râles are the most important, and when taken in connection with the history, the most significant.

In some instances the breathing may be decidedly cavernous, or even amphoric. This is due to excessive dilatation of a bronchus.

On the unaffected side there are exaggerated respiratory murmurs.

The heart necessarily shows changes. There is accentuation of the pneumonic second sound and, in advanced conditions, murmurs and the signs of displacement and dilatation.

X-RAY DIAGNOSIS, see Part VII.

Diagnosis.—The presence of chronic cough, mucous or muco-purulent expectoration, together with the physical signs mentioned and the history of the persistence of these symptoms for a long period, points to the presence of fibrosis of the lungs. A diagnosis, however, cannot be made until fibroid tuberculosis has been excluded.

The first point in the differential diagnosis is to distinguish the disease from tubercular fibrosis. The history and symptoms of both may be apparently identical, but in tuberculosis the disease is more apt to be bilateral, while in non-tubercular fibrosis it is rather more likely to be unilateral. This, however, is too indefinite a feature to form a substantial base of differentiation, the only positive point is the presence or absence of the tubercle bacilli in the sputum. The fact should not be overlooked that in fibroid tuberculosis several examinations are often necessary before the bacillus is detected, hence very careful consideration should be given each case and frequent examinations should be made. It is important also to note that cases of long standing may assume a decided hectic from retention of bronchial secretions in old bronchiectatic cavities, yet remain free from tubercular infection.

Prognosis.—The duration of the disease may cover a period of many years, even twenty or more. Death, however, often occurs from some intercurrent affection, as impairment of the general death renders such probable. The fact that the patient may become infected by the tubercle bacilli should not be forgotten. When the disease runs its course, the patient after many years of semi- and actual invalidism succumbs either from the cardiac complications or from general weakness as mentioned.

Treatment.—The management of cases of pulmonary fibrosis must be on general hygienic principles. The nutrition must be maintained at the highest possible standard. A little wine or whiskey with dinner is often advisable in those of advanced years. The patient should live in the open air as much as possible, dress warm and avoid cold and damp and the changes of weather. Whenever possible the winters should be passed in a warm or mild, dry equable climate. If bronchial symptoms are prominent, the iodide of arsenic or the iodide of potassium may prove of some service, especially if there are acute exacerbations. When fetid bronchitis or bronchiectasis develops the treatment should be according to the plans considered for these conditions.

PNEUMONOKONIOSIS.

The derivation of pneumonokoniosis from πνεύμων, the lung; κονία, dust, explains its meaning. It is applied to that morbid condition of the lungs which arises from the continuous inhalation of large quantities of dust.

Ætiology.—The disease is one of occupation and is due to the irritating effect of dust particles upon the lung tissue. According to the nature of the irritant it has been variously designated, thus anthracosis, when caused by coal dust, siderosis, when from the dust of iron, chalicosis, from minerals in general, byssinosis, from cotton fibres, etc. Colloquially it is called stonecutters' phthisis, knife-grinders' phthisis and millers' phthisis.

The conditions which favor the development of the disease are deficient ventilation and the quantity as well as the quality of the dust inhaled. The first is strikingly illustrated in mining and the second in grindstone manufactories. Mining has the additional injurious factor of deprivation of sunlight, which necessarily tends to lower the vitality.

Morbid Anatomy.—The lung is discolored according to the nature of the inhaled irritant. In miners, it is black; in workers in iron oxide, it is red. The pigmentation may be dense and uniform or variagated and mottled. Fibroid degeneration is the most markedly characteristic change. The ciliated epithelia and phagocytes which are normal to the respiratory mucous tract are engaged extensively with inhaled particles. The cilia push the dust-laden mucus to where it can be removed by coughing. The mucus and alveolar cells take up the dust particles and, unless the latter are very abundant, prevent their entering the lung tissue. When the foreign material is excessive, it escapes these cells and penetrates the mucous surfaces where it meets the connective tissue, which has the power of retaining large numbers of dust particles. In the occupations under discussion these natural resistive forces are overwhelmed and the lymph channels are entered, the particles being carried to the lymph nodules surrounding the blood-vessels and bronchi to the interlobular septa beneath the pleura, and by the large lymph streams to the substernal, bronchial and tracheal glands. In the latter instance they are prevented from reaching the general circulation by the stroma cells of the follicular cords of the gland structure.

In cases of anthracosis deposits of coal particles have been discovered in the liver and spleen, showing that they sometimes enter the general circulation.

Under the influence of a constant and abundant inpouring of the dust particles, hyperplasia of the connective tissue is excited and the process of fibrosis commences.

The starting-point is usually in the peri-bronchial lymphatics and in the early stages the changes are confined to these localities. In time the hyperplasia of the pulmonary connective tissue becomes general. The walls of the alveoli and interlobular septa show new connective tissue formation, while bands of fibrous tissue may be observed crossing the lung in various directions. The natural elasticity of the lung is much diminished and emphysema results.

Patches of dense, airless tissue, surrounded with dark, grayish crepitant tissue are found in the lung, especially in anthracosis, in the centre of which a bronchus is found. These patches are areas of fibroid broncho-pneumonia. They vary in size from a pea to a hazelnut. Sometimes they coalesce and form large indurated masses.

In addition to these changes chronic bronchitis plays a very important part and the pathological changes incident to it must also be taken into consideration. Emphysema also constitutes a more or less constant accompaniment. Bronchiectasis likewise results.

The bronchial glands are pigmented and enlarged and the pleura is thickened with adhesions. The heart may be secondarily affected, being hypertrophied and dilated.

In some instances the fibroid areas may undergo softening and form suppurating cavities with ragged walls. These cavities are usually small.

It is important to bear in mind that a tubercular process may become ingrafted upon that of pneumonokoniosis, as the latter naturally tends to place the lungs in a condition favorable for the reception of the bacillus.

Symptoms.—The clinical manifestations of pneumonokoniosis are essentially those of chronic bronchitis and emphysema and later of pulmonary fibrosis. Cough is one of the early symptoms, likewise dyspnœa, which as an accompaniment of emphysema may be asthmatic and paroxysmal. In cotton workers sneezing may be an early symptom.

The expectoration in the earliest stages is mucoid, later it becomes profuse and purulent and when there is bronchiectasis, fetid. In some cases it may be a bronchorrhœa.

In the case of coal miners it is black; in those who work in cotton white; thus varying according to the occupation. In workers in stone, gritty particles are sometimes found.

As the disease progresses, there is debility and loss of flesh. Night-sweats, fever and diarrhœa also develop as the result of absorption of septic matter from the retained pulmonary secretions or tubercular infection.

Dropsy may also develop, consequent upon obstruction of the pulmonary circulation and cardiac dilatation.

The course of the disease covers many years. A cough and some impairment of the general health may be present for a long period without other marked symptoms.

PHYSICIAL SIGNS.—The physical signs do not call for separate discussion, being those of chronic bronchitis, fibrosis of the lung and emphysema.

Prognosis.—While the protracted course of the disease permits patients suffering from pneumonokoniosis to live many years, the ultimate outlook is decidedly unfavorable, unless the injurious causal occupation ceases to be engaged in. It is notable that men engaged in the occupations under discussion seldom live beyond middle age. In the early stages, when the pathological changes have not gone far, if the patient is placed under favorable hygienic surroundings, the prospect is fair for the arrest of the disease.

Diagnosis.—The diagnosis scarcely requires special comment. Given the symptoms of chronic bronchitis, pulmonary fibrosis and emphysema, in a person constantly inhaling a dust-laden atmosphere, there can be no difficulty in recognizing the condition.

Treatment.—The first essential is obviously removal to proper hygienic surroundings, with abundance of pure fresh air and sunshine. The nutrition should also be maintained at a high standard. As far as remedies are concerned, those mentioned in the treatment of chronic bronchitis, emphysema and bronchiectasis should be considered.

ABSCESS OF THE LUNGS.

SYNONYMS.—*Suppurative Pneumonitis, Metastatic Abscess, Embolic Septic Pneumonia, Septic Pneumonia.*

Abscess of the lungs signifies, as in other localities, infiltration of a portion of the tissues with pus and white blood-corpuscles, and subsequent sloughing and dissolution of its connection with the surrounding structures.

Ætiology.—Predisposition is recognized in depraved states of the system from any cause, such as prolonged illness, chronic alcoholism, old age or diabetes.

The exciting cause is obviously pyogenic bacteria, especially the streptococci and the staphylococci, which gain entrance either by inspiration or through the blood channels.

Pulmonary abscess differs from abscess in other parts in that the surrounding tissues are often the seat of preëxisting disease. Thus there may be abscess following some other morbid condition of the lungs, as well as abscess in lungs previously healthy. The most frequent causes fall under the first head and include croupous, desquamative and aspiration pneumonia and tuberculosis. While croupous pneumonia does not very often terminate in abscess of the lungs, a larger proportion of cases arise from it than from any other one cause. Less frequently desquamative pneumonia may run into abscess. In aspiration pneumonia a foreign body may be drawn in to the bronchi, where it lodges and sets up a pneumonic inflammation which subsequently breaks down and suppurates.

In pulmonary tuberculosis, when large caseous masses have undergone rapid softening, abscesses may form.

Abscess formation in lungs previously healthy occurs in infectious embolism, and in connection with suppurative conditions, notably puerperal sepsis, gangrene, sloughing bed-sores, suppurative joints, abscess of the mediastinal glands, glanders, abscess of the liver, malignant pustule and caries and necrosis of any bone, especially those of the thorax.

Pulmonary abscess may likewise arise from contiguity, as rupture into the lungs of an abscess of the liver or mediastinum, or from extension of empyema. Rarer causes are traumatism, perforation from carcinomatous ulceration and pressure from thoracic aneurysm.

Morbid Anatomy.—Pulmonary abscess may be single or multiple. Its seat in about 80 per cent. of all cases is in the lower lobes. Those of pneumonic origin are usually single; those the result of general sepsis are multiple. In some instances there is a purulent infiltration of the blood-vessels and interstitial tissue of a certain part, but more frequently there is a circumscribed area of purulency. The size in the latter case may vary from a small walnut to that of an orange. The walls of the abscess are irregular and the contents purulent, rarely necrotic. In cases of long standing the walls may be fibrous. When near the pleura empyema usually results, although sero-fibrinous pleurisy may also occur. Pulmonary abscess may sometimes rupture into the pleural cavity.

Symptoms.—The presence of abscess in the lung is not always attended with clearly-defined symptoms. During the formation of the suppurating process the temperature remains high. Later it usually assumes a septic course. When abscess follows croupous pneumonia the course of the latter is usually characterized by irregularity and symptoms of asthenia, followed by improvement at the period of crisis, and afterward by an exacerbation of unfavorable symptoms in the form of rigors, fever, sweats and dyspnœa.

The sputum is of great diagnostic value. It is purulent and usually yellow, less often greenish or brownish in color. It emits a sweetish or fetid odor, but is by no means so offensive as in the case of gangrene of the lungs. On microscopic examination particles of elastic tissue fibres of the lung are seen. This is of great diagnostic importance. Leucocytosis is present.

PHYSICAL SIGNS.—The physical signs are those of consolidation before the discharge of the pus, and afterwards those of cavity in addition to consolidation.

Diagnosis.—The presence of elastic tissue fibres from the lung, copious purulent sputum and the constitutional symptoms of purulent infection and leucocytosis are the essential features upon which the diagnosis rests.

In the instance when infectious embolism is suspected and its possible source recognized, the presence of pain in the side, with symptoms of purulent infection and quick and superficial respiration, is strongly suggestive of pulmonary abscess.

The development of pleuritic friction murmurs in association with abscess of the lung must be regarded as indicating that perforation is imminent or has taken place.

Bronchiectasis might under some conditions be confused with abscess of the lung, but its sputum rarely contains elastic fibres, and that only when the bronchi are ulcerated.

A pulmonary abscess cavity may be distinguished from one of tuberculous origin by the presence of bacilli in the sputum, and in the instance of abscess associated with tuberculosis by the fact that the cavity of abscess is in the greater number of instances in the lower lobes.

Prognosis.—The outlook is unfavorable, although in the instance of a single abscess the prospects of recovery are sometimes quite fair. Abscess from embolism and multiple abscesses are very grave, also those associated with empyema and hepatic abscesses which discharge into the lung.

Treatment.—Support the patient by the administration of highly nutritious food and stimulants and in every way endeavor to counteract the great drain upon the system. Alcoholic stimulants are always necessary. Antiseptic inhalations, such as creasote, eucalyptus and carbolic acid, as mentioned under Tuberculosis, may prove of service in promoting the cough and as deodorants. No anodynes should be used to allay the cough. Generally constitutional remedies, such as hepar sulphur., silicia, arsenicum, arsenici iodidum, lachesis, quinia and echinacea should be administered according to individual indications.

When the abscess is near the surface of the lung surgical interference in the form of pneumonotomy for the evacuation of pus should be considered. When associated with empyema, resection of the ribs will be indicated and should be performed without delay.

Indications for operative interference are difficult to specialize. It may be stated, however, that the presence of secondary empyema points to the evacuation of pus in all cases of pulmonary abscess, also in cases uncomplicated with empyema with no prospect of evacuation by the bronchial tubes.

GANGRENE OF THE LUNGS.

Pulmonary gangrene is necrosis and decomposition of lung tissue. The process may be either circumscribed or diffused.

Ætiology.—In all instances it is a secondary affection. Three factors enter into its causation. First, predisposition; second, a primary disease; third, the bacteria of putrefaction.

Predisposition is manifest in all conditions which tend to depress the vitality, such as chronic alcoholism, diabetes, Bright's disease, faulty nutrition either through malassimilation or the vicissitudes incident to poverty.

The diseases which are liable to develop pulmonary gangrene when engrafted upon a constitution undermined by debilitating influences as above mentioned are as follows:

(1) Croupous pneumonia.

(2) Aspiration pneumonia, especially when the foreign body itself undergoes decomposition.

(3) Bronchiectasis and tuberculosis, where the contents of putrid cavities induce gangrene of the surrounding tissues.

(4) Pulmonary embolism, either simple or infectious. In the first instance the embolus may cause gangrene by occluding the vessel

and cutting off the blood-supply, in the latter it may carry with it the putrefaction germ from some other area. This may occur in sepsis of all kinds, local sloughing and suppurative conditions.

(5) Thrombosis of the pulmonary artery. This is an unusual but possible cause, and has occurred in infectious fevers, such as typhus and typhoid.

(6) Ulcerative processes, such as cancer of neighboring organs, as the stomach or œsophagus, or cancer of the lung itself.

(7) Pressure from aneurysm of the aorta, or tumor of the mediastinum.

(8) General infections, such as typhus, typhoid, measles, glanders, septico-pyæmia and snake poisons.

(9) Ulcerative processes of the mouth, pharynx, larynx and trachea. These may prove the source of infection by inhalation of septic germs from their secretions.

(10) Wounds of the lung. This may be caused by direct perforation or by indirect laceration and contusion of the lung tissue.

While the diseased conditions, which may prepare the way for infection of the lungs by the putrefactive bacillus, are many, it is only in a small proportion of such instances that pulmonary gangrene results.

The third causal factor and the specific cause itself is the putrefaction bacillus. The identity of this micro-organism has not yet been established with any degree of certainty. Various bacilli have been described by different investigators as the source of the lesion. The staphylococcus aureus and streptococcus pyogenes have been pointed out as the cause by one observer, a special micro-organism has been demonstated by another, and still another maintains that "various kinds of micro-organisms" are the probable cause. With such conflicting statements it is safe to conclude that the specific bacillus has not yet been recognized.

Morbid Anatomy.—In the circumscribed form the process consists of one or more areas of gangrene with the line of demarcation between the gangrene and non-gangrenous tissues clearly defined. The seat of the process is usually in the lower lobe of the lung. The right lung is more frequently involved than the left, and the periphery more than the central portion. Under this form gangrene of embolic origin is included.

The necrosed area ranges in size from one-third of an inch to an inch and a half in diameter. The process may consist of a fresh mortification which is soft, pliable in consistence and greenish-brown in color, or a firm eschar of greenish-brown or blackish formation with the tissues more moist, more compact than those of the normal lung, and at the same time harder. In appearance this formation is like an eschar on the skin following the burn of caustic potash.

On section the involved areas exude a foul smelling fluid. The lung surrounding the gangrene is congested, inflamed and often consolidated, and the air-cells are filled with the products of the decomposing lung tissue. There is also surrounding the involved area a marked degree of œdema. As the process advances the necrosed mass disintegrates and is expelled, leaving a ragged, shreddy cavity containing a putrid greenish fluid.

Vessels may be eroded and copious hæmorrhages occur. The adjacent veins may become occluded by infection and emboli may detach themselves and set up infectious processes elsewhere. The pleura may likewise become implicated and give way and pneumopyothorax result. The secretions cause intense irritation of the bronchial mucosa, but the bronchial tubes resist the necrotic process longer than any other pulmonary tissue. Ultimately, however, they succumb. In the rare instances where recovery takes place, the cavities formed by the gangrenous processes develop a lining wall of connective issue and contract to a greater or less degree.

The diffuse variety is rare. It may occur after lobar pneumonia or occlusion of a branch of the pulmonary artery. The latter is very unusual. The process may also arise secondarily to the circumscribed variety. The greater portion of a lobe or entire lung may be involved, but the process is less intense, sometimes becoming dry. The tissues are moist, breaking easily, as in post-mortem engorgement with serum. The color is lightish or dark green or almost black. The involved tissues become soft and pulpy ; a putrid mass yielding a grayish-green foul fluid. In some portions of the lung the almost healthy tissue merges gradually into the gangrenous area, in others there is a line of inflammation and engorgement dividing the two. Spots of hæmorrhagic infiltration may be observed.

Microscopical examination shows the presence of fragments of necrosed tissue, great quantities of bacteria of various kinds, notably the staphylococcus pyogenes and streptococcus aureus, also free fat, fatty crystals, changed blood and detritus.

Symptoms.—There is the history of some preceding disease or condition which more or less influences the symptoms which accompany those peculiar to pulmonary gangrene. The latter are very striking and consists of an unusually profuse and horribly offensive sputum of a dirty greenish-yellow or brownish color with a tendency to separate into layers, viz., the first or bottom layer consisting of a thick sediment like pus of a greenish-brown color, above this an albuminous, almost serous, layer of a greenish color, and an upper layer of greenish-yellow froth. Cough is more or less constant, arising from the irritating qualities of the secretions and the accompanying bronchitis. Hæmoptysis is frequent. It may be slight, the blood being mixed with sputum, or copious consisting of pure blood. The patient usually lies on the affected side. Dyspnœa may not be marked, except when the patient attempts to exert himself when it may become excessive.

Constitutional symptoms are profound, there is great depression of general strength and vitality. There is fever, sweats, irregular pulse and the symptoms peculiar to sepsis. Fever may not, however, be at all excessive, running from 102° to 103° F., or it may be absent. There may be gastro-intestinal irritation from the ingestion of poisonous matter. Emboli, pleurisy and pneumothorax are all possible complications. The association of abscess of the brain with pulmonary gangrene should also receive consideration.

PHYSICAL SIGNS.—There are no distinctive physical signs. There are the usual signs of consolidation, softening and excavation according to the stage of the disease. Areas of consolidation rapidly followed by excavation are very suggestive. When the areas of gangrene are deeply situated often the only signs discernible are bronchial râles.

Diagnosis.—The diagnosis depends for the most part upon the extreme fœtor of the sputum in association with the presence of necrosed masses of lung tissue and elastic tissue fibres, the physical signs of rapidly forming cavities and the symptoms of sepsis and systemic prostration.

Pulmonary gangrene may be confused with bronchiectasis, fetid bronchitis and abscess of the lung with fœtor. From bronchiectasis, which may be attended with fetid sputum separable on standing in layers, pulmonary gangrene can be distinguished by the more rapid and more profound prostration, the presence of necrotic masses of lung tissue and of elastic tissue fibres in the sputum (the latter is only present in bronchiectasis when the bronchi are ulcerated), the greater fœtor and frequently by the history of the preceding primary disease. From fetid bronchitis it may be differentiated by the sputum, which does not contain elastic tissue fibres and does not separate into the characteristic layers and is less foul. From abscess of the lungs with fœtor, by the peculiar character of its sputum as described; that of abscess is distinctly purulent.

Prognosis.—The outlook is exceedingly grave. The diffuse variety is invariably fatal. The circumscribed in a few instances has been attended with resolution. The disease usually runs its course in a week or less, although some cases may cover a much longer period. Death by exhaustion and sepsis is common. A fatal termination may also occur from hæmorrhage, pneumothorax, embolism or peritonitis.

Treatment.—Support the patient by an abundance of nutritious food, such as rich broths, milk, etc., given at short and frequent intervals. Stimulants should be administered freely in the form of brandy or whiskey. When the local trouble can be definitely recognized incision and drainage are recommended, as they have been successful in a number of instances. Local antisepsis is useful in allaying the fœtor, and may be used in the form of inhalation of carbolic acid, thymol, iodine, creasote, bromine, eucalyptus, etc. The mouth may also be sprayed. These measures, however, can have no effect in retarding the disease.

As far as drugs are concerned their utility is negative. Arsenic, echinacea, lachesis, and the internal antiseptics all have been recommended and results claimed from their administration.

SYPHILIS OF THE LUNGS.

Pulmonary syphilis may be either congenital or acquired. The former is the usual type, the latter is rare.

Morbid Anatomy.—The congenital variety may be either diffuse or circumscribed. The former is a pneumonic process and occurs in the still-born or in children who live but a short time. Generally such children are born prematurely. Two forms of pneumonic syphilis are recognized, the white pneumonia and the gumma. White pneumonia (pneumonia alba, fœtal pneumonia) is a more or less lobar consolidation. On section the lung is light gray or almost white in color, smooth, shining and airless; microscopically the walls of the alveoli are seen to be thickened and their cavities distended with cells, showing a tendency to undergo fatty degeneration. In some instances degeneration does not take place and the proliferation epithelia of the alveoli are crowded within the alveolar lumen. The blood-vessels show proliferation of the adventitia and intima or may be entirely obliterated.

In the circumscribed variety the process is that of gumma and is similar to that condition when the disease is acquired. Gumma may also be associated with the diffuse form.

Acquired syphilis of the lungs generally appears four or five years after infection. The pathological changes may be those of gumma or fibrosis or it may assume the form of an ulcerative process constituting what has been called syphilitic phthisis. While gummata as stated may be associated in the diffuse variety of infants, they are of more frequent occurrence in adults. The root of the lungs beneath the pleura is their most frequent seat, but they may be scattered throughout. Generally their number is limited. Their appearance is that of brownish bodies, grayish to yellowish in color, surrounded with more or less fibrous overgrowth which often radiates into the lung tissues. They vary in size from a cherry to a hen's egg. They may undergo necrosis in their centres and form cavities. Sometimes their contents may be discharged through the bronchial tubes or undergo absorbtion and a cicatricial contraction result.

Under the microscope the appearances ordinarily observed in gumma are seen, with more or less surrounding infiltration and thickening and hyaline degenerative changes in the blood-vessels.

The interstitial or fibroid form is that condition where the changes are similar if not identical to those which take place in pulmonary fibrosis. It is therefore an interstitial pneumonia characterized by the presence of a fibrous change. The process begins as a rule at the seat of the lungs and radiates throughout the pulmonary tissues towards the pleura. In some cases the process spreads from the pleura inward. In others again it may be so diffuse that the appearances presented is like that of pneumonokoniosis; it is, however, distinguishable from it. Bronchiectasis may be observed. Microscopic examination shows an increase of inter-alveolar and inter-lobular connective tissues which form broad bands or meshes.

The air-cells are crowded upon or obliterated, while their epithelia show the presence of desquamation. Gumma may be associated with these changes which seem to be more frequent in the right lung.

A broncho-pneumonic variety and lymphatic forms are mentioned by some writers. The former implies an acute syphilitic inflammation of the lung which would seem possible. The latter are said to be characterized by enlargement of the bronchial glands and infiltration of the lymphatics leading to the lungs. These lymphatics are distended with a creamy white fluid.

In the phthisical variety the lung breaks down as in tubercular infection.

Symptoms.—The disease may manifest itself in several ways; there is, however, nothing pathognomonic in its symptoms.

Gumma may be attended with cough, paroxysmal in nature, and with scanty expectoration, pleuritic pains, absence of fever, no emaciation and the physical signs of consolidation.

When the pathological processes are of a fibroid nature the symptoms are those of pulmonary fibrosis. Again when the process is ulcerative the disease may assume a course similar to tuberculosis of the lungs, with the symptoms ordinarily attending that lesion. Such cases are rare. Many diagnosed as syphilitic are doubtless tuberculous.

Diagnosis.—A positive diagnosis is always difficult and rests mainly on the results of treatment. A clear history of infection is obviously of very great importance. Bronchiectasis and pulmonary fibrosis may be regarded as of syphilitic origin when there are associated active syphilitic lesions or old cicatrices.

The presence of consolidation and cough, with the history of syphilis and absence of hectic, suggests gumma.

In those varieties which simulate pulmonary tuberculosis the diagnosis rests on the results of sputum examination.

Prognosis.—In the congenital variety infants usually live but a few days. In the acquired form the prognosis depends upon the extent of the lung involved and the general condition of the patient.

Treatment.—The treatment, it is needless to state, should be anti-syphilitic, mercury and potassium iodide being the cardinal remedies.

NEOPLASMS OF THE LUNGS.

The lungs may be the seat of carcinoma, sarcoma and enchondroma. Of these new formations, carcinoma is the most frequent, but it is by no means common, sarcoma is rare, while enchondroma is very unusual.

Ætiology and Morbid Anatomy.—The origin of cancer of the lung does not differ from that of the disease in other organs. It may be either primary or secondary, the latter constituting by far the larger proportion of cases. Heredity and age appear to exert the same degree of influence as in other cancerous affections, with the exception that according to German observers pulmonary cancer occurs at a much earlier period of life, ranging from twenty upward, the majority of cases being under forty.

Primary cancer has been observed more often among men than women, while the secondary variety seems rather more frequent among females, but the contrary is claimed by some writers.

Primary cancer is more frequently encephaloid, less frequently epithelioma and rarely scirrhus. Sarcoma, enchondroma and osteoma of primary origin also occur.

Primary pulmonary cancer appears to originate in the epithelia of the alveoli, in that of the bronchial mucous membrane and in the endothelia of the deep pulmonary lymph vessels. It is of interest to note that it has been found in association with pulmonary tuberculosis.

Traumatism is mentioned by Aufrecht of Madgeburg-Altstadt, who quotes Georgi and others as citing instances where heavy blows upon the chest appeared to be the direct cause.

Primary pulmonary cancer is generally a diffuse cancerous disease of at least one entire lobe of the lung. In addition there may be some slight enlargements of the mediastinal glands. The disease generally extends from the hilum of the lungs or attacks the

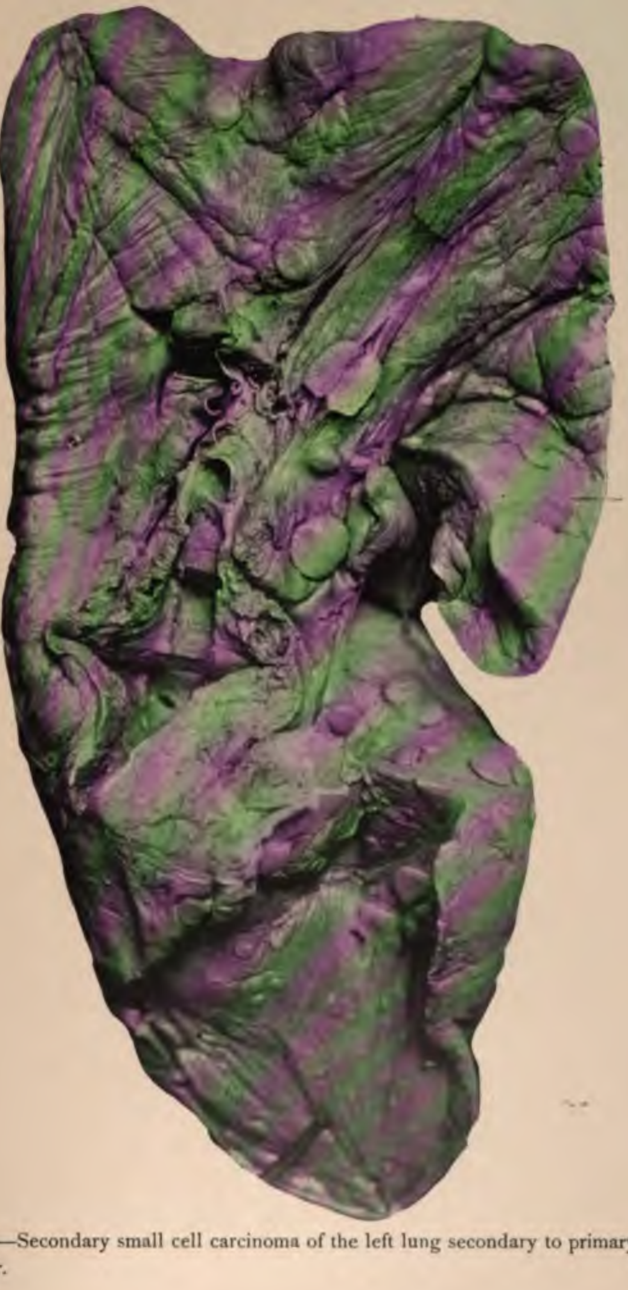

FIG. 52.—Secondary small cell carcinoma of the left lung secondary to primary cancer of the liver.

greater part of one lobe, or the whole lobe, and in some instances the entire lung. The other lung generally remains free. If the upper lobe is the seat of disease the process extends to the apex.

In most cases the disease advances along the bronchial vessels, rarely along the pulmonary. The bronchi often undergo changes, the smaller becoming constricted and the larger occluded, terminating it may be in blind sacs in the cancerous masses.

Secondary cancers of the lungs may be encephaloid, scirrhus, epithelioma, colloid, melano-sarcoma or enchondroma. They arise either from metastasis or contiguity. A large number of cases are those secondary to cancer of the breast in females. Many again arise secondarily in cancer of the alimentary and genito-urinary tracts. These do not, as a rule, attain so large a size as those of primary origin, for generally the system is greatly impaired by the primary disease, which does not permit of much development of the growth.

Generally the disease makes its appearance in the form of small nodules at different points in the lung at the same time. These enlarge by involving the adjacent lung structure, with the result that a large portion of the lung may become implicated. The nodules also coalesce and form large cancerous masses. When the disease is diffuse and involves the greater portion of the lobe, the centre of the diseased mass may undergo softening.

The changes in the lung structures in both forms of the disease naturally induce retention of secretions in the bronchi, sanguineous extravasations and hæmorrhages.

The bronchial and mediastinal glands become infiltrated and enlarged, the pleura studded with cancerous nodules and more or less inflamed. There is also effusion, either sero-fibrinous or hæmorrhagic. The tracheal and cervical glands show enlargement.

Microscopical examination reveals the preservation of the walls of the alveoli which are thickened and changed from the stroma of the neoplasm. The alveoli themselves are filled with large nucleated nuclei, generally spheroidal, but sometimes polygonal.

Symptoms.—The symptoms are inconstant, variable and often without definite characteristics. There is cough at first attended with mucoid expectoration and later with current-jelly-like sputum. There may also be hæmoptysis. Pain may be excessive, especially when the pleura is involved. In some cases the cancerous mass may give rise to pressure symptoms, causing dysphagia, dyspnœa and aphonia. Pressure upon the pulmonary veins may cause œdema, congestion of the lungs and hæmorrhage. Cachexia is usually present.

PHYSICAL SIGNS.—The physical signs are those of consolidation and bronchitis, and are in no way distinctive. It will be helpful to recall in this connection the pathological processes of the cancerous formations, viz.: Primary cancer originating in the alveoli occupies a portion or the entire lobe or even the whole lung. Cancer originating in the larger bronchi occupies a portion of the lung near the hilum and extends outward. Secondary cancer is isolated or consists of disseminated nodules throughout the lung.

In some instances the heart may be displaced.

X-RAY DIAGNOSIS, see Part VII.

Diagnosis.—The diagnosis of primary cancer of the lungs is difficult and can only be reached by the history of the case and an exploratory puncture. Secondary cancerous disease of the lung may be suspected when, in association with cancer elsewhere, cough, pain and pulmonary consolidation develop.

Pleural effusion may easily be mistaken for cancer of the lung. A point of difference is that the dulness over the lower lobe in the latter instance extends higher up. This, it is evident, is not conclusive and recourse must be had to the exploratory needle. In fact, it may be said that in all primary cancers of the lung and in many of secondary origin no definite diagnosis can otherwise be made.

Prognosis.—The outlook is utterly hopeless.

Treatment.—Notwithstanding the futility of any endeavor for the arrest of cancer of the pulmonary variety, remedies will be necessary for the relief of symptoms and palliation of the patient's condition. The bronchitis, dyspnœa, cough, hæmoptysis, and especially pleurisy and empyema, will demand attention and the remedial agents for treatment of each of these conditions as discussed elsewhere should be employed.

HYDATIDS OF THE LUNGS.

Hydatids of the lungs is the presence in the lung substance of the larvæ of the tænia echinococcus, a parasite which in certain localities infests dogs, and to a less degree wolves, jackals and sometimes sheep.

Ætiology and Morbid Anatomy.—Discussion of the development of the echinococcus in general is not within the scope of this article. It will suffice to recall that the parasite is especially prevalent in Iceland and in parts of Australia and is also observed, but less frequently, in Manitoba and Argentina. In this country and in Great Britain it is very rare.

In Iceland, where hydatids are a comparatively frequent cause of death, certain classes of the inhabitants allow their dogs to literally eat and sleep with them.

The usual mode of infection is by drinking infected water, although it may occur through direct contact by caressing and fondling the infected animals.

When the egg of the echinococcus is taken into the stomach the embryo is quickly freed. It then either burrows through the intestinal wall and enters the peritoneal cavity or some of the muscular structures, or it passes into the portal circulation or into the systemic vessels. The common course is that of the portal circulation, hence hydatids are more common in the liver than elsewhere; after the liver the lungs are the most frequent seat.

Having located, the embryo loses its hooklets and gradually becomes metamorphosed into a cyst, consisting of a thick, elastic outer wall and a granular inner wall, containing a transparent non-albuminous neutral fluid. After the death of the hydatid, this fluid becomes turbid and albuminous.

When the cyst has attained an age of about five months, it has a diameter of about half an inch and echinococcus heads begin to bud from its walls. In time, these buds develop into cysts, with similar structures. The second or daughter cysts are at first attached to the lining membrane of the parent cyst, but soon become detached. From these in turn other cysts form.

Hydatid cysts of the lung may be either single or multiple, generally the former. The right lung is more frequently affected than the left and the lower lobes than the upper.

The presence of the cyst in the lung substance naturally presses upon the surrounding tissues, causing irritation and inflammation. In cases of long standing there may be induration with the formation of a fibrous capsule, but generally there is no encapsulation.

Suppuration may arise from compression, leading to abscess, which may discharge in any direction. As a result of abscess there may be cavities or gangrene.

There is always danger of rupture of the cyst. This may take place into the bronchi, the most frequent locality, or into the pleural or pericardial cavities. The cyst may also undergo a suppurative process.

Symptoms.—When the cyst is small, symptoms may be absent or not at all striking. Later they become decidedly manifest, but often without any characteristic feature. In the earlier periods of the affection, there may be only a slight dry hacking cough ; later there is mucous expectoration. There may be hæmoptysis either slight or profuse, usually the former. Pain is not present unless the pleura is implicated. Fever and dyspnœa are also absent.

If the cyst dies and suppurates, the symptoms become those of pyæmia in conjunction with those of pulmonary abscess.

When rupture occurs, the symptoms naturally vary according to the seat of the same. If into the bronchi, there is a profuse watery expectoration, which on examination will reveal the presence of the hooklets, small daughter cysts and fragments of the cyst's membrane. There may also be hæmorrhage. While the bronchi are the most favorable points for rupture to take place, such an accident may be attended with death from suffocation.

If the cysts rupture into the pleural cavity there is severe pain, dyspnœa and collapse. If into the pericardium, sudden death.

After rupture there follows fever and putrid expectoration with pigments of the hydatid. There are also chills, fever, sweats and emaciation, and the general symptoms of hectic.

PHYSICAL SIGNS.—The physical signs are those of consolidation, the locality of which varies. If near the surface and of sufficient size, the cyst may cause some prominence between the interspaces. Again should the cyst be near the surface there may be present on palpation the so-called "hydatid thrill."

After rupture there may be the signs of a cavity. Other changes may be observed, as œdema, râles and pneumonic consolidation, according to the effect of the irritation on the surrounding lung tissue.

Diagnosis.—The history of exposure to contaminated dogs, the physical signs of a solid area in the lungs with vague symptoms may be regarded as suggestive. Conclusive diagnosis, however, can only be reached by demonstrating the presence of the echinococcus hooklets in the sputum, or the discovery of hydatids elsewhere in the body.

Hydatid cysts should never be aspirated, as the results are often disastrous.

Prognosis.—Of cases allowed to go on to spontaneous rupture, if death does not occur at the time, about one-half recover. Cases which undergo operation generally recover.

Hæmorrhage and especially fetid expectoration and œdema and suppuration are unfavorable.

Treatment.—Removal of the cyst by operation is the only method of treatment. Medicines are of no avail.

ACTINOMYCOSIS OF THE LUNGS.

Actinomycosis of the lungs is a chronic infectious disease caused by the streptothrix actinomyces or ray fungus.

Ætiology.—The ray fungus enters the human organism presumably by alimentary or respiratory tract from infection from cattle. The mode of infection, however, is a matter of some doubt, inasmuch as the fungus has not yet been discovered outside the body of either man or cattle.

Morbid Anatomy.—The lesions are usually unilateral. The primary infection assumes the form of bronchitis, broncho-pneumonia and miliary-like bodies and nodules. The miliary bodies, the central portion of which in the earlier stages are yellowish-white, resemble those of tuberculosis, but consist of groups of the fungi surrounded with granular tissue. In more advanced stages the foci disintegrate and form cavities containing pus, fatty detritus, fatty granules, disintegrated cells and masses of radiating threads of the actinomyces.

The tissues between the foci are more or less inflamed, indurated and thickened. Connective tissue may form which results in a grayish, slate-colored, callous, airless mass. In some cases again the process may push near the surface and involve the pleura, giving rise to inflammation, exudation and adhesions. After the costal pleura has become implicated, the cellular infiltration, pus, fatty degeneration and granular tissue may push forward, penetrate between the ribs through the walls of the chest and form a sinus. In some instances the suppurating mass may break into the mediastinum, pericardium or heart or through the diaphragm into the abdominal cavity.

Similar processes may be observed in other parts of the body with implication of the bones. There may also be metastatic abscesses in different parts.

Symptoms.—The onset is insiduous and the course of the disease gradual, but steadily progressive. General loss of strength, anæmia and cough are the earliest symptoms. The patient loses flesh, and the cough, which at first is dry, is accompanied later by a muco-purulent and fetid expectoration containing yellow granules which may be recognized as the ray fungus. There is also fever and sweats and the symptoms common to suppurative processes. As the condition advances emaciation becomes marked. Later the chest wall shows evidences of change, there is bulging of the affected side, the tissues of which become swollen and inflamed, and an abscess makes its appearance. In some instances the disease processes pushes in other directions as mentioned.

The course is about one year. In its earlier periods it may suggest pulmonary tuberculosis. In some instances it may simulate typhoid fever.

PHYSICAL SIGNS.—*Inspection* shows anæmia and emaciation, especially in advanced stages, when there is also often bulging of the chest wall at the point of involvement. *Palpation* adds little to situation. *Percussion* generally shows dulness and symptoms that simulate pleuritic effusion. *Auscultation* may be indefinite or may show bronchial râles and bronchial respiration.

Diagnosis.—The diagnosis depends upon the recognition of the ray fungus in the expectoration, or in the discharge of an abscess if one has formed.

Prognosis.—Death usually occurs from exhaustion, pyæmia, amyloid degeneration or embolism. A few recoveries, however, have been recorded.

Treatment.—No special line of treatment has been effective. Iodide of potassium in full doses has been recommended. Other remedies are argent. nitricum, acid. carbolicum and mercurius corrosivus. The patient should receive nutritious diet and should be supported by every possible measure against the great drain and tendency to sepsis. Theoretically the fungus should be removed by an operation, but its situation in the lung precludes such interference.

ASPERGILLUSMYCOSIS OF THE LUNGS.

SYNONYMS.—*Pneumonomycosis.*

Aspergillusmycosis of the lungs is a rare disease due to infection by the fungus, aspelligus fumigatis. The expression "pseudo tuberculosis" has been applied to the condition, but is misleading and inappropriate.

Ætiology.—The disease occurs among those who work in grain, flour, meal, hair and seeds, which have become infected with the spores of the fungus. It also affects the lower animals. Hair-combers in Paris found that birds could not live in their homes more than two weeks, dogs not more than two months, while cats were not susceptible.

Morbid Anatomy.—The pathological changes resemble those of pulmonary tuberculosis. Infection results in a similar tubercle. Inflammatory changes and necrosis follow. Two forms are described, the inflammatory and the abortive, but the rarity of the disease scarcely render practical a detailed description. If such is desired the reader is referred to the work of Dr. Renon, *Etude sur L'Aspergillose chez les Anemaux et chez L'Homme*, Paris, 1897.

Symptoms.—The clinical manifestations of infection by the aspelligus are not always constant. In some instances there is apparently little harm done and few symptoms arise, while in others the symptoms and general course of the infection strongly resemble those of tuberculosis. There is general decline of health, loss of appetite, morning vomiting, weakness and progressive emaciation. Cough is present, at first dry and paroxysmal, but later attended with expectoration which is frothy in the earlier periods of the disease, but later becomes green, purulent and sometimes blood streaked. There are also night-sweats and an evening rise of temperature. Dyspnœa becomes marked. Pleuritic complications may develop, and in the advanced stages profuse hæmorrhages may occur, also œdema of the lower extremities. In some cases the symptoms may set in with severe hæmorrhage.

Secondary aspergillusmycosis is recognized. It occurs with bronchiectasis, emphysema, broncho-pneumonia, tuberculosis, infarction from hæmorrhage secondary to valvular disease and carcinomata.

PHYSICAL SIGNS.—The physical signs are those of bronchitis and consolidation generally at the apex.

Diagnosis.—The diagnosis of the condition and its differentiation from pulmonary tuberculosis depends upon the recognition of fungus in the expectoration, that is the mycelium or small rounded white or yellowish-white body which occurs in great numbers. In suspected cases the sputum should also be examined for tubercle bacilli in order to eliminate the possibility of that infection. Aspergillusmycosis of the lungs and tuberculosis, however, may both be present.

The occupation of the patient in suspected cases will be of assistance in diagnosis.

Prognosis.—The prognosis is serious. In some instances the disease is arrested, restoration being attended with fibroid changes of the lung similar to those of arrested tubercular disease. It is said that the disease may be of long duration, sometimes continuing for six or eight years.

Treatment.—The patient should be removed from the source of infection and live in the open air as much as possible, following the same general lines as in pulmonary tuberculosis. The general strength should be built up and maintained by highly nutritious diet and general tonics. The cough and hæmoptysis should be treated as outlined elsewhere.

Renon found that animals with the disease when given potassium iodide and arsenic linger longer and therefore recommended these remedies for man.

DISTOMA WESTERMANNI.

This is a parasite found in the lungs of cats, tigers, dogs and man. It is of Asiatic origin. It may infect man by inhalation. The symptoms are similar to those of pulmonary tuberculosis. The treatment consists of the inhalation of antiseptics, especially creasote, general tonics, life in the open air and plenty of sunshine. The affection is rare.

THROMBOSIS OF THE PULMONARY ARTERY.

The formation of a thrombosis in the pulmonary artery may occur in stasis of the blood, when death is gradual, in certain pulmonary diseases, such as pneumonia, tuberculous cavities, fibroid induration of the lung, atheroma of the aorta either gouty or syphilitic, vitiated conditions of the blood, and in those cases of embolism of the pulmonary artery where life is prolonged, and the process follows secondarily as the result of stasis of the general circulation.

The symptoms are severe dyspnœa, cyanosis and evidences of profound embarrassment of the circulation similar to those produced by embolism of the artery. Treatment is without effect. Full doses of morphine should be given hypodermically, and the patient kept under its influence.

EMBOLISM OF THE PULMONARY ARTERY.

When a large embolus passes into the circulation and lodges in one of the main branches of the pulmonary artery it gives rise to a pathological condition and symptoms materially different from those which result from occlusion of a small branch of the artery in the lung substance itself. While the source of large emboli includes all those morbid conditions mentioned in the discussion of Pulmonary Infarction, their usual origin is disease of the endocardium and walls of the right side of the heart associated with stasis and passive congestion of the pulmonary circulation.

The effect of a large embolus is the total or partial arrest of the circulation of the lung. Death is usually instantaneous.

If the obstruction is not complete, or a narrower but still quite large branch is obstructed and death not instantaneous, the patient is suddenly seized with an agonizing pain in the chest, great sense of oppression and terrible mental anguish. The countenance is pallid at first, later cyanosed, the jugular veins distended, the pupils dilated and the eyeballs protruded. The heart action is tumultuous and irregular. Sometimes there is delirium, convulsions and unconsciousness. The patient may linger from a few hours to several days.

In those cases in which death is not immediate, morphine hypodermically in sufficient doses to keep the patient under its influence should be administered.

SECTION III.

DISEASES OF THE PLEURA.

PLEURISY.

SYNONYM.—*Pleuritis.*

PLEURISY is inflammation of the pleural membrane. It may be either local or general and affect one or both pleuræ.

Pleurisy is classified according to the sequence of its appearance as primary or secondary, according to its duration, as acute, subacute and chronic, and according to the pathological changes as fibrinous, sero-fibrinous and purulent. All these varieties share the same ætiology.

While recognizing the several forms and stages of the disease there are many instances where one merges into another and where the dividing line between stages and types is not well defined.

The term pleurisy is very ancient. It was used by Hippocrates to signify all conditions attended with pain in the chest walls, especially those of a severe character. It was also mentioned by Celsus and by Galen, who better defined it. Æctaeus was the first to speak of it with any degree of precision. These writers regarded the disease as seated in the layer of the pleura over the ribs and other parts. Later writers viewed the process as principally implicating the pulmonary pleura. Sydenham and Morgagni thought that both the lungs and the pleura were affected. Pinel was the first to establish a definite distinction between inflammation of the pleura and that of the lungs. Laennec cleared the situation by proving that pleurisy was a distinct disease entity, showing that the inflammatory process could commence in the pleura and be limited there or could extend to the lungs.

Ætiology.—PREDISPOSING CAUSES: There are many factors which act as predisposing or exciting causes of pleurisy. These are chiefly those which were regarded as constituting the ætiology as accepted before the discovery of the bacteriological origin of the disease.

Exposure to cold was long looked upon as the principal cause of pleurisy of sudden development in previously apparently healthy persons, but modern pathology regards it as simply an exciting cause, holding that the primary source of the disease is microbic infection, generally tubercular. There are, however, a certain proportion of cases of this type which cannot be traced to tubercular infection. Whether or not future investigation will be able to demonstrate that these also are tubercular remains to be seen. Many of such pleurisies may be regarded as manifestations of mild infection of the lungs or bronchial glands, or at least evidence of a predisposition to tuberculosis. There are others, again, which suddenly appear and resolve without any apparent after-effect. Such cases are doubtless of a rheumatic origin. Therefore at the present time it is well to hold a conservative position in regard to the acceptance of the view that all so-called idiopathic cases of pleurisy are tuberculous.

Another group of exciting causes is acute and chronic inflammation of the lungs, especially when the process is near the surface. Here the pleuritic inflammation is secondary. Thus pleurisy is common in pneumonia, tuberculosis, abscess and gangrene. Traumatism is also an important ætiological factor, such as fractures of the ribs and heavy blows on the chest.

The acute infections are another class of diseases which are prone to favor the development of pleurisy, more especially endocarditis and sepsis. Acute articular rheumatism is the mildest of the group. Intensity of rheumatic inflammation as in the instance of peri- and endocarditis is not necessarily a criterion as to the liability of pleuritic inflammation. In measles, notwithstanding its tendency to pulmonary complications, it is not common. Typhoid fever is also seldom attended with pleurisy, even when the lungs are involved. In chronic nephritis, degeneration of the myocardium and hepatic cirrhosis pleurisy with effusion is of frequent occurrence.

Inflammation of the pericardium, peritoneum or mediastinal structures are often attended with pleurisy. Under these conditions the disease arises either by extension through the parenchyma or by way of the lymph channels. Likewise it may arise from ulceration of adjacent structures, namely, the ribs, stomach, œsophagus or mediastinum.

Still another group of predisposing causes are constitutional diseases of almost any type, profound disturbances of nutrition, old age and debility after prolonged illness and after hæmorrhage. In the latter the pleurisy is liable to be of thrombotic origin.

BACTERIOLOGY.—The actual cause of pleurisy in all its forms according to modern pathology is micro-organisms or their irritant products. There are three varieties of bacteria which are chiefly concerned in the process; these are the tubercle bacillus, the diplococcus lanceolatus or pneumococcus of Fränkel and the streptococcus pyogenes. The rôle played by these micro-organisms in the various manifestations of the disease has been classified by Netter as follows:

(1) *Pleurisy Due to Tubercle Bacillus.*—In regard to this variety the following observations are presented: (*a*) A tendency to pleurisy in tuberculous subjects; (*b*) the association of tuberculosis with sero-fibrinous pleurisy; (*c*) pleurisy arising suddenly in previously healthy persons has been shown by autopsy or subsequent history to be tuberculous; (*d*) those who recover from this form of pleurisy frequently subsequently develop tuberculosis. According to Bowditch, thirty-two out of ninety cases of pleurisy died of pulmonary tuberculosis. Of one hundred and thirty-one cases of all varieties, Osler found on post-mortem thirty-two to be tuberculosis.

Bacteriological examinations of the exudate either by cover-slip preparations or by cultures are almost always negative. This is due to the very small number of the bacteria. When large quantities of the exudate are taken the test is often positive. This was demonstrated by Eichhorst, who took 15 c.c. of the exudate and was able to demonstrate the micro-organism in 65 per cent. of his cases. Le Damany used still larger quantities, doses of 10 to 15 c.c., in some instances giving the animal as much as 300 c.c. of the exudate, with the result of proving the tuberculous nature of all but eight out of fifty-five cases of pleurisy.

(2) *Pleurisy Due to Diplococcus Lanceolatus.*—In this group are found the sero-fibrinous, purulent pleurisies secondary to croupous pneumonia and many of the cases of so-called primary empyema in

children. Netter showed the presence of the pneumococcus in thirty-two out of one hundred and nine cases of purulent pleurisy. Other observers have reported similar results. The exudation in these cases is usually purulent and the prognosis more favorable than in those forms due to other micro-organisms.

(3) *Pleurisy Due to Streptococcus.*—This variety is encountered in empyema of adults and is generally secondary to inflammatory processes principally in adjacent structures. It occurs in association with streptococcus pneumonia and broncho-pneumonia, pulmonary abscess and gangrene and wounds and inflammations of the chest and neck. In some instances it may arise by extension from the peritoneum or by general infection through the blood. The process is essentially septic and is the most serious of all forms. In children streptococcus pleurisy is frequent with diphtheria, scarlatina, measles and occasionally with whooping-cough. Netter in one hundred and fifty-nine purulent exudates found the streptococcus in fifty-one.*

While it has been demonstrated that pleurisy is generally caused by one of the three varieties of micro-organisms as mentioned, this must not be taken to imply that the bacteriology of the disease is uniform, for the infection may be mixed. Again, numerous other bacteria are found, namely, the staphylococcus pyogenes aureus in association with suppuration in other parts of the body, Friedlander's bacillus, diphtheria bacillus, colon bacillus, micrococcus tetragenus and occasionally the typhoid bacillus. In pleurisy due to the typhoid bacillus, the process according to Charrin, Rogers and others may arise independently of disease of the lungs and may be either serous, hæmorrhagic or purulent; generally it is the last.

Bacteriological examination of the exudate of pleurisy with rheumatism and cancer have so far been sterile.

Notwithstanding the general acceptation of the microbic nature of pleurisy there are cases of so-called idiopathic origin which have not been brought within the realm of bacteriology.

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* Diseases of the Pleura, O. Rosenbach, M.D., Nothnagel's Encyclopedia of Practical Medicine, Philadelphia and London, 1903.

1. Acute Fibrinous Pleurisy.

SYNONYMS.—*Plastic Pleurisy, Dry Pleurisy.*

This variety of pleuritic inflammation is characterized by mildness, localization of the inflammatory process and absence of serous effusion. Two forms are recognized, the primary and the secondary.

Ætiology.—While the same general ætiology and bacteriology of pleurisy is shared as stated by all varieties of the disease, it may not perhaps be amiss to dwell briefly upon the casual influences in their relationship to the different types of the disease, bearing in mind that the same factors in one case may result in fibrinous pleurisy, while in others they may be followed by the serous form.

(1) Primary fibrinous pleurisy may follow exposure to cold or wet, and includes those mild cases generally regarded as idiopathic, many of which are erroneously so classified, for in a large proportion there is some diathetic taint, such as rheumatism or gout. Many again are either tuberculous or are characterized by a tendency to tuberculosis.

Pleurisy of this type is more likely to be encountered in the winter and spring and is more frequent among males than among females.

(2) Secondary fibrinous pleurisy is more frequent. It arises by extension from some acute or chronic disease of the thoracic organs and is often observed in croupous pneumonia, less frequently in broncho-pneumonia and more rarely still in infarct, abscess, gangrene and cancer. It may likewise occur with acute articular rheumatism as a secondary process, either as part of the general infection or by extension from pericardial inflammation.

In hepatitis, fibrinous pleurisy may arise by extension, hence in pleurisy of the right side this possible origin must be borne in mind.

Among chronic diseases it is most frequent in pulmonary tuberculosis, where in fact it is almost always present to a greater or less degree. As it is possible for fibrinous pleurisy to be the primary manifestation of infection in pulmonary tuberculosis, the lesion stands in a double relationship to tuberculous infection. It may also appear with peritonitis where infection may take place through the lymphatics, and in chronic Bright's disease and in chronic alcoholism as a complication.

Morbid Anatomy.—The disease is almost always unilateral. The changes are usually localized, although they may become general. Sometimes they are confined to reflections of the pleura separating lobes of the lung. The first change is congestion, the membrane becomes dull, due to clouding of the epithelia and slightly rough or granular from the distortion of the vessels and swelling of the endothelial and of the connective tissue cells. After a few hours' exudation the second step in the process appears. The surface of the membrane now becomes covered with a thin whitish fibrinous layer; a delicate film which can be readily scraped off leaving beneath the congested and inflamed pleura. The exudation is the result of a coagulation necrosis of the endothelia.

It is usually more pronounced on the costal than on the pulmonary pleura. When on the latter it is generally found only in the region of the interlobular fissures. Later it increases in thickness, becoming at the same time yellowish in color. With this increase the surfaces of the pleuræ become agglutinated and present a rough, shaggy appearance, like that of two pieces of buttered bread which have been pressed together on their buttered surfaces and then separated. On account of the friction between the two pleural surfaces in dry pleurisies of very decided types the exudate may become very thick.

The more fluid portion of the exudate may ultimately become absorbed, while the more solid part through the presence of embryonic cells develops connective tissue and blood-vessels, with the result that the opposing surfaces of the pleura become adherent. The adhesions are delicate and cellular at first, but subsequently become dense and firm. They vary in density and extent, but are usually more abundant at the apex and root of the lungs. Where the movements of the lung are more extensive, as at the base and margin, there are usually no adhesions; but in their place there are often ribbon-like bands formed of the more elastic portion of the exudate, the result of the movements of the lungs in respiration which draws the latter out in this form.

Microscopic examination shows the exudate to be composed of granular masses, fibrils and flakes of fibrin with more or less round-cell infiltration. Beneath the exudate the membrane is seen to be thickened owing to the proliferation of the endothelial cells, some of which are observed to be separated.

In mild types of the disease connective tissue formation and adhesion do not occur, as the disease process does not go to such a length. Under these conditions the products of inflammation undergo fatty change and absorbtion and finally in favorable cases the pleural surface becomes completely restored.

Thus it will be observed that the pathological changes in fibrinous pleurisy pass through these stages, congestion, exudation and resolution. Again, while in favorable cases the greater portion of the fibrinous exudate may be absorbed, some may remain causing thickening of the pleura. In some instances the fibrinous exudate may undergo cheesy degeneration.

When repeated attacks of pleurisy occur, as is frequently the case in pulmonary tuberculosis, if the process is not sufficiently extensive to cause adhesions, the pleural surface becomes thickened and opaque in spots from proliferation of the connective tissue cells, as a result a good deal of thickening of the pleura follows.

Symptoms.—The onset of fibrinous pleurisy varies widely. The subjective symptoms may be severe, very mild or negative. Generally there is a sharp lacerating pain in the side, usually located a little below the nipple. The pain is aggravated by respiration and movement of the body. The patient therefore assumes a fixed position, at the same time restraining as much as possible the movement of the chest walls in breathing. The respirations are short, jerky and somewhat accelerated. The pain continues for about two or three days, when it subsides leaving a sense of soreness.

There may be an initial chillness, sometimes, though rarely, a severe chill like that attending pneumonia. There may also be severe pains in the back.

The temperature is in no way typical, ranging up to 103° F., but generally about 101° F. The pulse is usually from ninety to one hundred and twenty and is small or soft. Cough is present; it is usually dry and hacking. There is expectoration only in severe cases and those accompanied by bronchitis. In these varieties the temperature rises to 104° F. There is also general prostration and more marked constitutional symptoms.

The mild forms of the disease may be at first of such undefined or negetive character that the subjective symptoms scarcely attract attention. The patient may complain of an uneasy sensation in the side and malaise, but continues perhaps to perform his usual duties. Secondary manifestations of dry pleurisy are usually of this type. Many, again, are without any symptoms at all and the lesion is only discovered at autopsy.

PHYSICAL SIGNS.—*Inspection* in characteristic cases shows the jerky, hurried respiration and the endeavors of the patient to restrain movement of the affected side when breathing, and consequent deficient expansion.

Palpation in the earlier periods of the disease simply confirms inspection, later when exudation is well established it gives tactile fremitus. A friction rub on palpation is mentioned by some observers. *Percussion* is generally negative, but there is slight dulness detected when the exudate is considerable.

Auscultation, when exudation has taken place, shows the presence of a rubbing, grating friction-sound, crepitating in character, most intense at the end of inspiration, but heard on both inspiration and expiration and intensified by deep breathing. These sounds are audible for about two days, then disappear. In exceptional instances the exudate may be sufficiently extensive to compress the lung, in which case the affection may be attended with bronchial breathing and possibly may be mistaken for croupous pneumonia.

Diagnosis.—The recognition of fibrinous pleurisy depends mainly upon the presence of a sharp pain in the side, with the signs on auscultation of the rubbing, grating friction murmur, the latter being the essential feature.

Pleurodynia may simulate dry pleurisy; both are attended with a suddenly appearing sharp pain in the side, and both may also be excited by exposure. Intercostal neuralgia may likewise present, in a less degree, some of the appearances of dry pleurisy. Both these affections, however, are readily distinguished from pleurisy by the absence of friction murmurs on auscultation. In intercostal neuralgia there are also painful pressure points (Valleix's pressure points) and in some instances herpes develops.

Prognosis.—Recovery from the immediate attack is the rule in from three or four days to three weeks. The ultimate prognosis is by no means so favorable, for one attack predisposes to another, with the result of causing pleuritic thickening, adhesions and interference with lung expansion and subsequent organic changes in the lung itself. The presence of adhesions may also form the source of long continued pain, which is generally intensified by exercise, breathing and change in the weather. In time, however, the gradual stretching of these adhesions may afford relief. The possibility that dry pleurisy may be of tubercular origin must always be taken into consideration. When fibrinous pleurisy arises secondarily in connection with nephritis, hepatitis or peritonitis, it may act as a terminal complication.

Treatment.—The first indication and the first demand of the patient is the relief of the pain. This can usually be accomplished by the local applications of heat or cold and the continual administration of the indicated remedy. These measures generally render the administration of morphine unnecessary. Locally the ice-bag or Leiter's coil is recommended by some. It is generally suited to the early stages only. Heat is usually more agreeable. It may be used in the form of the hot water bag, flannels wrung out in hot water or hot fermentations. The latter may be employed in the form of a flaxseed meal poultice over which a half or an ounce of laudanum may be poured. Sometimes mustard poultices act well; if made by mixing the mustard with boiling castor oil, blistering will be avoided. Strips of adhesive plaster tightly drawn from the sternum to the spine so as to restrain the movements of respiration are of decided benefit and may be all that is necessary. It is very important that the plasters should be applied at the end of expiration, otherwise they are ineffective.

The patient should remain in bed; the diet should be light and the bowels relieved.

Aconitum will prove of service if the case is seen very early, especially when there is much fever with restlessness.

Bryonia is the principal remedy, it is indicated at any stage of the disease, but more especially after exudation. Severe stitch-like pains in the side, aggravated by motion, desire of the patient to support the side, or to lie upon it, short rapid respiration, white-coated tongue, thirst for large quantities of water at long intervals and torpor of the bowels are its principal indications.

Æsclepias tuberosa, when there is a dry hacking cough, little expectoration, sharp shooting pains, relief from bending forward, especially in dry pleurisies of pneumonia and pulmonary tuberculosis.

Kali carbonicum after bryonia is one of the most important drugs for fibrous pleurisy. Sticking pain more especially on the left side, stitches of pain in region of the right scapula on breathing, especially in the morning, dry cough and pain in the nape of the neck indicate its administration. Kali carb. is distinguished from bryonia by the fact that the indicating symptoms are aggravated by rest and by lying on the affected side, which is the opposite to those of the latter. Aggravation often after 3 A.M. is another guiding indication.

Scilla is indicated when the pain is so intense that there is difficulty in breathing. The cough is in this instance dry, hacking and irritating, it may also be paroxysmal and terminate in sneezing. There may be on the other hand free expectoration. Scillitin, the alkaloid, has proved more effective. It may be used in the second decimal trituration.

Sulphur is suggested when there are stitch-like pains in the chest extending to the back or left scapula when lying on the back, sensation of constriction of the chest, short, violent dry cough, debility and prostration.

Ferrum phos., hepar sulphuris, sabadilla, rhus tox. and arnica should also be considered. When gout or rheumatism is the cause the salicylates and colchicine will prove of benefit, especially the latter.

When convalescence sets in the patient should practice deep breathing to expand the lungs, if possible, and detach the remaining adhesions. If the pain continues after the subsidence of physical signs the constant electric current over its seat will often be attended with benefit.

2. Acute Sero-Fibrinous Pleurisy.

Acute sero-fibrinous pleurisy is that variety of pleuritic inflammation which is characterized by a greater degree of intensity, a more general involvement of the pleural membrane, and a more or less abundant effusion of serous fluid into the pleural cavity.

As in the fibrinous form two varieties are recognized, the primary and the secondary.

Ætiology.—The ætiology of sero-fibrinous pleurisy is identical with that of the fibrinous variety. The exciting and predisposing causes are the same as in the case of fibrinous pleurisy and bear the same ætiological relationship as to frequency. Probably the greater severity of the inflammation which characterizes the lesion is due either to a more intense virulence of the infection and more feeble resistive powers of the patient. However this may be, the relation of this form of pleurisy to microbic infection is much more clearly defined than in the fibrinous variety. *A large proportion of cases are tuberculous.*

(1) Primary sero-fibrinous pleurisy.—It is variously estimated that from two-thirds to three-quarters of all cases are tuberculosis. The old idea that sero-fibrinous pleurisy occurring in an apparently healthy person was simply an inflammatory process, which alone places the lungs in a condition favorable for subsequent infection, is now abandoned, as most of these cases are tubercular from the onset. Rheumatic infection is responsible for a certain proportion of cases.

(2) Secondary fibrinous-pleurisy.—One of the most frequent sources is secondary infection from pulmonary tuberculosis. The disease may therefore arise both primarily and secondarily from tubercular infection. The same two-fold ætiological relationship is manifest in regard to rheumatism where it may also arise secondarily in association with peri- and endocarditis with an acute articular rheumatism.

Pneumococcus pleurisy may be sero-fibrinous and secondary to pulmonary infection, usually pneumonic, rarely broncho-pneumonic. In the other acute infections it may also develop as a secondary complication. The typhoid bacillus of Eberth has proved to be an excitant of sero-fibrinous and purulent pleurisy.

Sero-fibrinous pleurisy may occur secondarily to peritonitis, nephritis, cirrhosis of the liver and in association with malignant growths of the thoracic viscera and liver.

Morbid Anatomy.—Sero-fibrinous pleurisy may be simply a further stage of the fibrinous form or it may be attended almost from the onset with a serous effusion. It is customary to describe the affection as consisting of three stages, viz., congestion and inflammation, effusion, and absorbtion. It is apparent in the instance when the effusion sets in very early that the dividing line between the first and second stages is not definable. Like the fibrinous variety, it is most always unilateral. Bilateral involvement is very rare.

The pathological changes of the first stage correspond to those which occur in fibrinous pleurisy. The second stage is characterized by the serous effusion into the pleural sac, that is, a fluid is formed in addition to the exudate on the pleural surfaces. The exudate consists of several layers of deposit with an abundance of inflammatory products. The amount of exudate and its proportion to the serous effusion is variable. In some instances it is scanty, in others free; in the latter instance it may appear on the pleural surface as a thick creamy deposit in layers as stated. In these cases the products of inflammation are abundant and there are whitish curd-like masses in the dependent parts of the fluid. The fluid varies in amount from a few minims to several pints. In color it is light yellow or yellowish-green, often opalescent, sometimes reddish from the presence of blood. It may be clear or cloudy from coarse shreds of fibrin, masses of cells or minute blood clots. Outside the body when left to itself it slowly coagulates, but coagulation may be hastened by heating. The specific gravity varies from 1005 to 1035; as a rule the specific gravity is greater in proportion to the amount of foreign matter present, such as blood, fibrin, etc. On standing a heavy sediment appears consisting of degenerated red blood-corpuscles in various stages of fatty degeneration and occasionally red blood-corpuscles in a good state.

Clinical examination shows that the fluid resembles blood serum containing albumin, cholesterin, sugar and peptone. According to Ewald no oxygen is found, but carbonic acid is abundant.

The fluid naturally seeks the lowest level of the pleural cavity. When the functions of the lungs, respiratory muscles and diaphragm are in good condition, however, the fluid level will be higher than would be supposed. This is owing to the aspirative influence of the lungs in respiration. It is important to observe in this connection that the surface of the fluid is never horizontal even when the amount of effusion is considerable and the lung compressed. The line being curved like the letter "S" as described in the Physical Signs.

The level and liberation of the fluid may also be modified by the presence of sacculations. These result from the more solid part of the products of inflammation, settling in such a manner that open spaces are formed in the solid masses of exudate which thus constitute isolated or communicating sacs, a condition termed sacculated or multilocar pleurisy. (*Vide* Special Forms of Acute Pleurisy.) The changes are the combined results of absorbtion, the presence of cells and additional exudation processes.

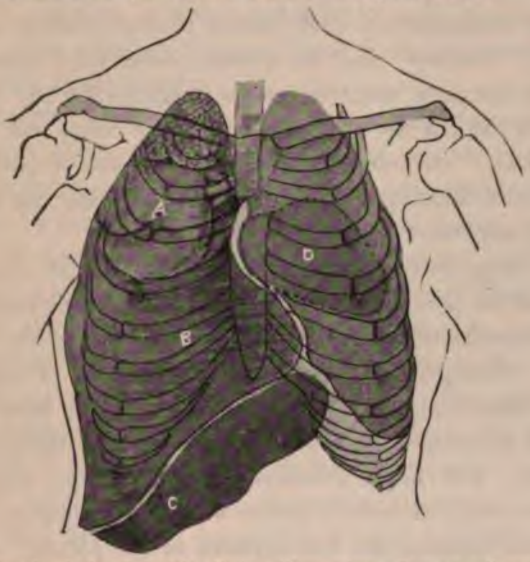

FIG. 53.—Sketch of pleurisy with effusion illustrating effect on adjacent organs. A, compressed lung, percussion gives dull tympany. B, fluid. C, depressed liver. D, displaced heart.

It is generally assumed that if the pleural cavity is only half filled the lung does not lose its contractile power. Large effusion causes changes which vary according to the amount of the fluid. When excessive the lung is pushed upward and back against the vertebral column and becomes atelectatic, being changed into a small mass, flat, airless and almost bloodless. While these changes in the lung are chiefly due to the pressure of the fluid, according to Strümpell the blood-vessels and fluid may absorb some air and thus contribute to the atalectatic condition. In addition to the mechanical effects upon the lung, the effusion exerts more or less pressure upon the mediastinum causing displacement of the heart. The mediastinum in addition loses the traction force of the diseased lung and in consequence is drawn by the other toward the healthy side.

When the heart is displaced the normal relation of the apex to the base is maintained even in extensive left side effusions. In some instances the apex may be elevated, while in others the organ may lie transversely, the right side being turned toward the anterior surface, so that the displacement of the organ is essentially displacement of the mediastinum itself rather than a rotation or twisting of the heart. Pressure may be exerted downward upon the diaphragm. On the right side this results in displacing the liver downwards and on the left the stomach, transverse colon and sometimes even the spleen. In some instances on account of strong prēexisting adhesions displacements of these organs may not occur, notwithstanding excessive effusion.

The third stage in the pathological process is that of absorbtion. The ability of the pleura to absorb the fluid exudates varies according to the intensity of the inflammatory process and the activity of the tissues. The greater the extent and density of the exudates the less the absorbing power of the pleura. The most favorable course is the absorbtion of the fluid exudates and the precipitation of the fibrin. The latter afterwards may undergo more or less organization into connective tissue with the formation of bands and adhesions between the two surfaces of the pleura. These adhesions are of varying extent.

The blood in both fibrinous and sero-fibrinous pleurisy, as a rule, does not present a condition of leucocytosis. This was shown by an observation of Morse in a series of two hundred and twenty-four separate blood counts from primary cases, in only thirteen of which was the number of leucocytes above ten thousand.* "The condition is valuable in two ways. If the physical signs are doubtful and there is no leucocytosis, the case is probably one of serous pleurisy and not of pneumonia or empyema. If serous pleurisy is present and there is marked leucocytosis, it is probable that a complication exists."

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* American Journal of Medical Sciences, November, 1900. Dr. J. N. Musser, Nothnagel's Encyclopedia of Practice, Philadelphia and London, 1903.

When extensive the functions of the lung suffer more or less serious impairment and the thoracic cavity itself may undergo a change from retraction of the chest walls. The thoracic muscles on the affected side may atrophy and the articulations of the ribs ossify. In rare instances the pleural sac may become entirely obliterated (synechia).

In other instances absorbtion may be limited and a certain amount of effusion remains indefinitely until the case is terminated by some associated lesion which in the majority of instances is pulmonary tuberculosis.

Symptoms.—As in the fibrinous variety extreme variability characterizes the early manifestations of sero-fibrinous pleurisy. While it is not possible to make any clearly definite classification, there may be said to be two general types, namely, those in which the onset is characterized by more or less activity of the symptoms, and those in which the early symptoms are of gradual development. In the former, like pneumonia, there is an initial chill which may be severe, and with it there is pain in the affected side, usually in the region of the nipple or on a line with the axilla. When the diaphragm is involved pain is referred to the abdomen and lower part of the back. (*Vide* Diaphragmatic Pleurisy.) The pain is of a catching, sharp, lacerating character, intensified by coughing and breathing, especially if the respirations are deep. Cough may be present without expectoration.

Gradual development of symptoms characterizes the greater proportion of cases of sero-fibrinous pleurisy. Even in healthy and strong subjects the first symptoms are often vague and indefinite, the patient experiences a feeling of weariness, slight difficulty in breathing, loss of appetite and debility. There are sensations of chilliness, more or less marked digestive disturbances and pallor. He also complains of some pain in the chest. At this stage if a physical examination is made a slight degree of dulness in the lower and lateral portions of the chest will be found. In another type the patient complains of chilliness, especially in the back, and pains of varying degree of intensity. There is also slight elevation of temperature at night and profuse perspiration. In cases of this variety sometimes the patient may not seek medical advice until the disease is well established and the effusion marked, especially is this likely to occur in those of the lower classes. Thus a patient may present himself complaining of general debility, anorexia, perhaps, pain and soreness of the side, perhaps none unless questioned, yet examination will show a temperature of 100° to 101° F., pulse accelerated and evidences of effusion in the pleural cavity.

The symptoms which attend the course of the disease like its onset are more or less variable and are best discussed in detail.

Fever, while generally present does not follow a typical curve, ranging from 101° to 102° or 103° F. Sometimes the temperature may be only moderately elevated, sometimes again there is a continued fever of 104° to 105° F. with intercurrent chills. This suggests purulent infection. The temperature usually rises as the effusion increases, the highest elevation corresponding to the period of greatest secretory activity. It falls gradually as the effusion is absorbed. A rise of temperature or a continued fever may therefore, in general, be said to denote increase of the exudate, a fall or irregularity of fever or a normal curve, on the other hand, to denote a diminution. This observation must be received with reservation as there obviously may be other conditions which must strongly bear upon the symptoms, especially in secondary forms. The duration of the fever is from one to three weeks, when it falls to normal, the physical signs remaining unchanged. At first the fall is in the morning, later in the evening, and as the case progresses the temperature curve becomes irregular with afternoon and evening elevations.

According to the observation of Peters,* the axillary temperature on the affected side is higher than that on the healthy.

Pain is a very important symptom. It varies in locality and intensity. It is usually referred to a point without the mammillary line in the fifth and sixth intercostal space, but may be as low down as the seventh. It may also be seated beneath the sternum and in rare instances in the axilla and beneath the clavicle, or it may radiate to both sides of the vertebral column, arms, shoulder and epigastrium. In children epigastric pain is more frequent. Some-

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* La France Medicale, May 4, 1878.

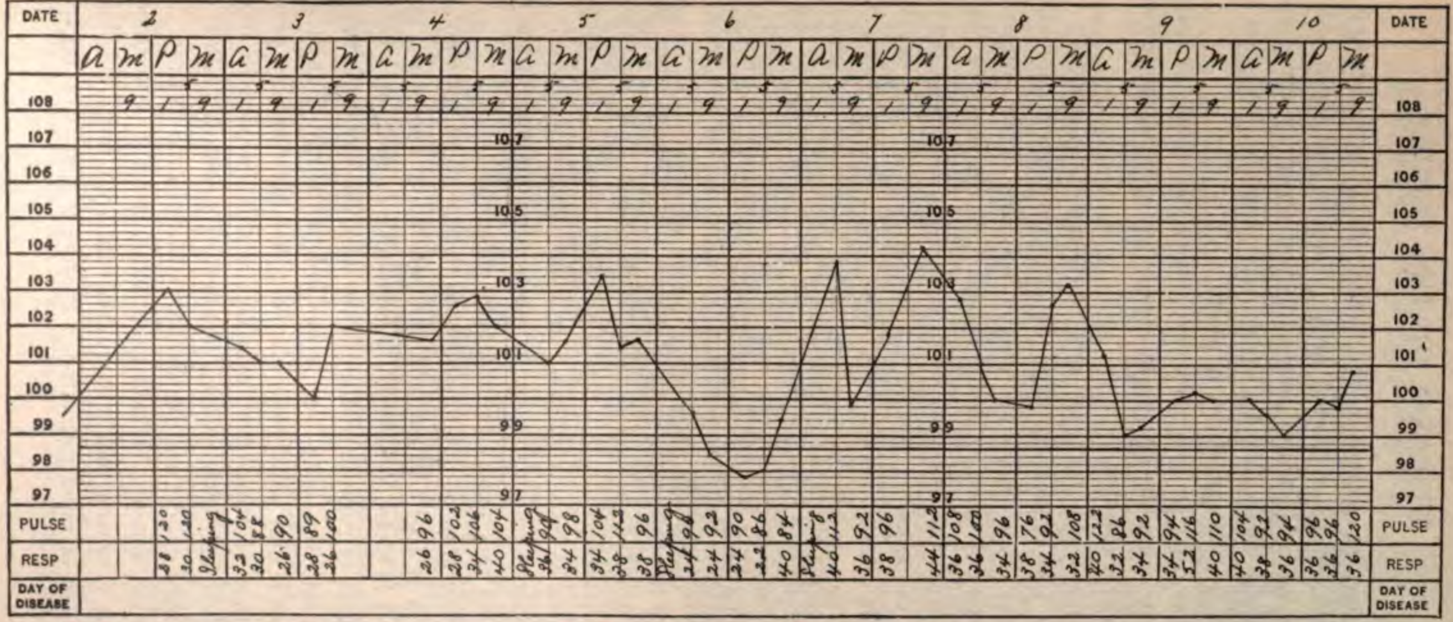

FIG. 54.—Temperature chart of a case of sero-fibrinous pleurisy with acute articular rheumatism. Patient aged 39 years. Metropolitan Hospital.

times it may be present on the sound side as well, where it is usually restricted to a limited area. Pain in the right side, back, shoulder or epigastrium points to involvement of the diaphragm. Occasionally there may be severe pain over the liver due to perihepatitis or pressure of the effusion.

The pain may be mild or severe, sharp, lacerating or cramp-like, or it may resemble intercostal neuralgia with sensitiveness along the vertebral column.

Accompanying the pain and following its subsidence there is more or less tenderness even on light percussion; sometimes the tenderness is excesssive the patient flinching at the slightest touch.

Dyspnœa is more or less constant. At the onset of the disease it arises from the pain and fever, later from the compression of the lung by the effusion, especially if the latter is of rapid development. When the effusion on the other hand has come on slowly, the lung may be very much compressed without causing dyspnœa, except when the patient exerts himself. In the acute stage of the disease at the commencement, the rate of respiration may reach forty or fifty. The breathing is superficial and short; later the rate falls to twenty-five to thirty.

Cough appears early in most cases. It is aggravated by sitting up and drawing a long breath which causes it to become painful and distressing. It is usually dry and hacking, but may be accompanied by expectoration. In some cases it is absent. Violent paroxysms of cough may occur when large amounts of the fluid are withdrawn too rapidly by operative measures.

Expectoration is not an essential symptom of pleurisy. When present it is slight in amount and mucoid in character, in rare instances it is blood streaked. Abundance of sputum, while usually a symptom of rupture of the pleura with consequent oozing of the effusion into the lung, may occur independently of such a contingency, as it may arise when the lung has been heavily pressed and a condition of temporary bronchiectasis is induced in one of the larger bronchi, within which masses of greenish-yellow purulent matter may collect and be discharged from time to time.

Cyanosis appears in those cases when there is pronounced dyspnœa and is consequently more frequent with large effusions, especially those of rapid formation. It should always be regarded as a grave symptom, as it indicates that the pressure of the effusion is seriously interfering with the oxygenation of the blood.

Pallor coming on rapidly with cyanosis in the course of the disease is suggestive of a hæmorrhagic effusion. When on the other hand it makes its appearance slowly it is usually independent of hæmorrhage.

The pulse in the earlier periods of the disease in uncomplicated cases corresponds to the temperature curve, later it is more especially influenced by the exudate. If the temperature ranges over 102° F. the pulse is usually to one hundred and ten to one hundred and twenty. In mild cases when the temperature is above 100° F. the pulse is from eighty to ninety. The more extensive the exudate the higher as a rule the pulse and the smaller its volume and lower its tension. In pronounced cases, therefore, the pulse is small in volume and low in tension.

Singultus is not an uncommon concomitant of severe types of the disease. It may also sometimes occur in mild cases, and in weak patients may prove a serious symptom. It implies implication of the diaphragm and is due either to irritation of that muscle or of the phrenic or pneumogastric nerve, more often the latter.

The urine does not manifest any characteristic change. It displays the same conditions as in other febrile disturbances. As the disease progresses the amount is lessened, this is due to a lowering of the arterial tension. A large exudate and a weakened heart are necessarily attended with direct mechanical effects, the result being diminution of the flow of urine, in part the result of the pressure of the effusion, which impedes the venous circulation, in part from the lessening of the respiratory movements and in part from the lowered arterial tension. Albuminuria may arise and present various grades of development.

When the effusion undergoes absorbtion rapidly there is an increase of urine.

The digestive tract is subject to certain disturbances to which allusion has been made. These will appear with varying degrees of intensity. Jaundice may be associated with right side pleurisy. It is not as a rule excessive.

When there is much fever and the symptoms are very acute, there is the thirst and anorexia common to such conditions. In cases of less active nature when the course is slower, there may be decided digestive disturbances and anæmia. Vomiting may be caused by the mechanical irritation of the exudate.

The position which the patient assumes is characteristic. The more intense the pain the more marked the tendency to lie upon the back or in a kind of diagonal position with the body bent. During the acute stage the patient lies generally on the well side, exceptionally on the affected side, sometimes pressing against it with the hands. Movement is avoided on account of pain. When effusion has taken place the patient lies on the affected side or back. When the effusion increases in amount the patient gradually evinces a tendency to lie upon the back or to sit up.

In cases which run a protracted course there may be great debility and emaciation.

Course.—The course of the disease is variable. It may be said to consist of a febrile and a non-febrile period. The first lasts for from one to three or four weeks and corresponds to the stage of inflammatory action, the latter is indefinite continuing for several weeks to as many months, and corresponds to the subsidence of the active process. Cases of rapid development usually run a rapid course. Those which appear with less pronounced symptoms follow a more protracted course. Resolution corresponds, being more rapid in rapidly developing cases and the reverse in those of insidious development. Generally the duration of the disease is anywhere from one to two months. In favorable cases the time required for the absorbtion of the fluid varies from several weeks to several months; generally it is very gradual. Extensive effusions may persist and the disease become subacute or chronic. In some instances the effusion becomes purulent.

Complications.—The most frequent complications are pericarditis and endocarditis which are more liable to occur in pleurisy of the left side. The possibility of the occurrence of embolism in the latter must be considered. Syncope and sudden death may occur when the effusion forms very rapidly, especially when there is fatty degeneration of the heart. The tendency of the disease to be followed by pulmonary tuberculosis to which allusion has been made, can scarcely be called a complication, as according to the views of modern pathologists, it is rather part of the infection.

PHYSICAL SIGNS.—The physical signs vary according to the stage of the disease.

First Stage.—At this period the physical signs are the same as those of the fibrinous variety.

Second Stage.—*Inspection* shows loss of expansion on inspiration. If the effusion is large there is increase in the size of the affected side with obliteration or bulging of the intercostal spaces—the latter is rare. The position of the heart shows more or less displacement. When the effusion is on the left side the organ may lie behind the sternum and be imperceptible or it may be pushed to or outside of the right mammillary line and elevated to the third and fourth interspaces. When on the right the apex is displaced to the left and upward, in some cases even as far as the axilla. The displacement is usually more marked when the disease is seated on the left side. Another important change is displacement of the liver downward; sometimes the spleen is also displaced. These displacements may simulate enlargement of the organs.

In some instances there is œdema of the skin on the affected side. This is more frequent in purulent effusion, but may occur with serous pleurisies when the effusion develops very rapidly.

Sometimes even in moderate effusion, more especially when there is marked sensitiveness of the surface, there is what might be called a want of perfect synchronism of the respiratory movements, that is, the expansion of inspiration and the contraction of expiration on the affected side are delayed.

Mensuration shows the affected side to be larger, usually by from one-half to one and a half inches. Due allowance should be made for the right side of the chest being normally somewhat larger than the left. It is important to measure the chest during expiration or while the patient is holding the breath.

Palpation confirms the signs observed on inspection and more definitely locates the position of the heart's impulse, but the most important sign is diminution or loss of tactile fremitus. The degree of diminution of fremitus usually is governed by the extent of the effusion; if the latter is slight the vocal fremitus is only lessened, if very extensive it is lost. Fremitus may persist in sero-fibrinous pleurisy when there are extensive bands of adhesions between the costal and pulmonary pleura and in infants, especially when crying. It is often less marked in women than in men.

Generally in effusion of moderate amount three zones of fremitus are present, that is, three variations. Over the lowest, fremitus is much diminished or absent, becoming more distinct as the next level is approached until at the third level normal fremitus is reached. The cause of these variations is apparent, the lowest zone corresponds to the situation of the greatest amount of fluid and the area of the greatest degree of retraction of the lung, the second zone to a smaller amount of fluid, the third to that portion of the lung free from the disease process.

It is important to observe that even in cases when the pleural cavity is well filled there may be an area of considerable size in the interscapular space where the fremitus is well maintained. This arises from the fact that the lung, although retracted, is not compressed and lies in close proximity to the walls of the chest. Another explanation offered is the presence of a large bronchus which is superficial. Before palpation is performed the patient should, if possible, sit erect and cough or take long breaths.

Percussion shows flatness which is absolute and which is an essential sign of effusion. It differs from the dulness of consolidation in being more positive and wooden in quality, and is attended with a decided resistance to the pleximeter finger. The extent of flatness on percussion varies with the amount of effusion and its line gradually rises as the effusion increases. When the patient is placed in the erect position the upper surface of the line of flatness is not on an even plane, that is, not on a level, but is higher posteriorly than anteriorly. When the effusion is moderately large this line commences at the lowest line posteriorly, passes upward and forward in a letter "S" curve to the axillary region whence it advances in a straight line, inclining downward to the sternum. This line is generally called "Ellis' line of flatness," or "Damoiseau's Curve." It is observable only when the lungs are in a fairly good condition, since by their elasticity they control the position of the fluid, and when the pleura is free from adhesions. When the effusion is excessive this curve flattens to a considerable extent. In some cases when the effusion is very extensive the line of dulness may reach the clavicle and extend laterally beyond the opposite side of the sternum.

The same rules in practicing percussion should be followed as in tactile fremitus. The patient should sit erect, take long breaths and cough. Most writers speak of change of position of the line of dulness on change of the patient's position. This, however, is not always obtainable. On the posterior surface two or three zones of dulness can be determined, a lower of flatness and a higher of normal note, or somewhat higher than normal. Between these zones there is one where this note is loud and more or less tympanitic corresponding to an area of retracted and relaxed, although not atelectetic, lung tissue. In the instance of very large effusion the entire posterior surface may be flat, with the exception of an area near the vertebral column in the interscapular space when the retracted lung is close to the chest walls. In the lateral walls two zones are present, one of a mild degree of flatness and one above when the note is moderately tympanitic. When the effusion reaches the interscapular space the latter zone becomes dull.

In the anterior wall a low pitched percussion-note is present just beneath the clavicle in the second intercostal space. Below, the note is dull. When the effusion has risen to the height of the fourth rib and compression has increased with consequent atelectasis of the lung, the percussion-note may be tympanitic even in the presence of the fluid. This phenomenon is called Skodaic resonance. It sometimes may also be observed posteriorly about the effusion line.

Another sign occasionally observed with large effusions is "Williams' tracheal tone." This is a high pitched note elicited by strong percussion just below the inner extremity of the clavicle. It is tympanitic in quality and changes with opening and closing the mouth. It is the result of vibrations coming from the bronchi and trachea through the compressed lung which has become permanently collapsed and retracted. Traube's space should also be examined. In this locality flatness may sometimes be detected even when the exudate is not excessive.

In estimating the amount of exudate its transverse direction must always be taken into account. For example, when retraction of the lung is limited owing to the presence of emphysema, adhesion, etc., the breadth or thickness of the fluid is obviously lessened and the same amount of effusion would rise to a higher level than if the lung was more compressible, thus the effusion may be of narrow breadth and reach to a high level.

Percussion should be practiced in regard to the displacement of the liver and heart. In effusion on the left side the dulness merges into that of the liver.

Auscultation during the stage of effusion shows that the pleuritic friction rub of the first stage has disappeared. The respiratory murmurs are variable. When the effusion is excessive they are very faint or entirely inaudible. In other instances bronchial breathing is present. Sometimes crepitant and subcrepitant râles are heard.

In large effusions three zones of ausculatory signs may be determined. The lowest, where the respiratory murmur is very feeble or entirely absent; a second, where there is more or less pronounced high pitched bronchial breathing; and a third or upper zone, where there is vesicular respiration. Bronchial breathing is generally more pronounced laterally and between the shoulders posteriorly than in the dorsal region.

As the effusion rises there may finally remain only a faint murmur at the apex. In some cases amphoric breathing and metallic tinkling may be heard over certain points in the interscapular space to the right and in the infra-clavicular fossa.

The varying respiratory phenomena on auscultation are accounted for by the progress of atelectasis and hepatization of the lung which follow as a result of the compression by the fluid; crepitant and subcrepitant râles may make their appearance, then disappear, and bronchial breathing of a high pitched character takes their place or the latter appears and is displaced by the former.

Associated with the bronchial breathing there is more or less bronchophony, the presence of which may be considered as a general indication of clearness of the bronchi; with a moderate degree of effusion in these cases the bonchophony may assume the peculiar bleating tones known as ægophony.

The sign of Baccelli must also be mentioned in this connection. This consists of the determination of whispered resonance. If such is present it was claimed by Baccelli that the exudate was serous, if absent, that it was purulent. This sign is not reliable, as sometimes whispered resonance may be absent in serous effusions and present in purulent.

Third Stage.—This period of the disease is characterized by the absorbtion of the exudate and a tendency to retraction. There is in consequence a gradual return to a normal condition. *Inspection* in many cases shows a variable amount of retraction of the chest walls with consequent displacement of organs. The retraction may be general or localized, the intercostal spaces may be more or less narrowed, the shoulders lowered and the nipple nearer the median line. The spinal column may be curved with the convexity toward the healthy side, there may also be some prominence of the scapula on the diseased side. In some instances, especially in children, there will remain sufficient expansive power in the damaged lung and thoracic wall to expand and overcome these tendencies to changes in the conformation of the chest. On the other hand the deformity may become permanent.

Mensuration shows a gradual decrease in the size of the chest on the affected side which finally becomes smaller than the other. *Palpation* gives a gradual return to normal fremitus from above downward. If there is much thickening of the pleura or adhesion, diminution or absence of vocal fremitus may persist for a long period or become permanent. *Percussion* also gives a gradual return to the normal. The flat note is succeeded by dulness. These changes naturally begin above and proceed downward. In the lower portion dulness is present for a long period. *Auscultation* discloses the reappearance of the respiratory sound which becomes gradually more distinct; when the fluid has disappeared to a considerable extent the roughened pleural surfaces may come together and give rise to pleuritic murmurs which may continue for many months after the absorbtion of the fluid. In some cases the lung remains permanently collapsed, with compensatory emphysema in the upper portion. With the absorbtion of the fluid the heart and liver return to their normal position.

X-RAY DIAGNOSIS, see Part VII.

Diagnosis.—Flatness on percussion, partial loss or absence of tactile fremitus and of the respiratory murmur are the essential physical signs of sero-fibrinous pleurisy, which when present with the history of symptoms, such as have been described, leave little doubt as to diagnosis; at the same time these signs are by no means absolute and several conditions must be eliminated by differential diagnosis. When a positive diagnosis is not manifest, exploratory aspiration should be employed in order to ascertain the condition. While the withdrawal of fluid is conclusive the failure to do so does not prove its absence. Failure to withdraw fluid notwithstanding its presence may occur when there is much thickening of the pleura, when there are thick fibrous bands at the point of puncture, when the needle is blocked with flocculent matter, and when the effusion is encapsulated. It may also occur when the effusion is purulent and thick.

The diseases which have proved a source of difficulty in diagnosis are croupous pneumonia, hydrothorax, pericarditis with effusion, intrathoracic growths, and hepatic abscess or cyst.

From croupous pneumonia sero-fibrinous is distinguished by the following points of difference :

SERO-FIBRINOUS PLEURISY.	CROUPOUS PNEUMONIA.
Onset characterized by chillness or general feeling of malaise.	Onset, sudden chill often very prolonged and severe.
Pain sharp and diffuse.	Pain similar, but generally more localized.
Cough usually dry.	Cough soon attended with expectoration.
Expectoration, if present, usually scanty and catarrhal.	Expectoration rusty and bloody, more or less abundant, shows the presence of pneumococci.
Fever usually moderate.	Fever high, declines from the fifth to the tenth day.
No crisis.	Crisis.
Systemic prostration moderate.	Systemic prostration severe.
Countenance pale and sometimes anxious.	Countenance usually flushed. Sometimes mahogany flush on cheeks.
Absence of herpes.	Herpes common on lips and face.
Leucocytosis absent or slight.	Leucocytosis usually decided.
Obliteration or bulging of the intercostal spaces or general distention of the affected side.	Absent.
Diminution or absence of tactile fremitus.	Increase of tactile fremitus.
Flatness on percussion.	Dulness on percussion.
Displacement of the heart and liver may occur.	No displacement of neighboring organs.
Line of flatness may sometimes be observed to change when the sac is partly filled.	Absent.
Diminution or complete absence of respiratory murmurs. Bronchial breathing may be present, but is diffuse and distant. Generally râles are absent.	Respiratory murmur always present. At first there are crepitant râles, later circumscribed bronchial breathing, later still the râle redux.

Sero-Fibrinous Pleurisy.	Croupous Pneumonia.
Vocal fremitus diminished or absent.	Vocal fremitus increased.
Ægophony—sometimes bronchophony.	Bronchophony decided.
In the early stages friction murmur.	Friction murmur absent.
Aspiration shows the presence of a serous fluid.	Aspiration yields simply blood.

It is important to note in this connection that the plugging of a large bronchus with secretions in pneumonia may cause the tactile fremitus to lessen or disappear. The harsh bronchial breathing and râles may also disappear under the same conditions.

Hydrothorax posesses the same physical signs as sero-fibrinous pleurisy, but is distinguished by the fact that it is usually bilateral and accompanied by decided evidences of renal or cardiac disease. Unilateral hydrothorax is possible in hepatic and cardiac disease if the venous flow from one pleura, generally the right in hepatic disease, is met with more than usual resistance. Under these conditions the diagnosis is difficult.

Double pleural exudates and the presence of an exudate upon which a transudation has been superimposed and the reverse are also very difficult to diagnose. The history of the case and the presence of renal or hepatic disease generally prove the main points upon which a true knowledge of the condition can be obtained.

According to Rosenbach and Pohl iodine or its salts when taken by the mouth are quickly recovered in large quantities in the transudate of hydrothorax, whereas in the exudate of pleurisy only a trace is found. These observers direct that a small amount of fluid be obtained by puncture and tested by the addition of fuming nitric acid and agitation of the fluid with chloroform. If the chloroform at the bottom of the tube is not colored red, pleuritic effusion is present, if red, the fluid in the pleural cavity is that of hydrothorax; the same observers claim that if a trace of red appears it is indicative of a complication of exudate and transudate.

Pericardial effusion if excessive may simulate that of pleurisy. The history of the case is an important help. In pericardial effusion there is frequently rheumatism and while dyspnœa is a prominent symptom and the heart-sounds are enfeebled, the heart itself is not displaced. Again there is flat tympanitic resonance in the posterior parts of the axilla and normal resonance at the base of the lung in the posterior and lateral positions of the chest. The pulse is small and compressible and the veins of the neck may become distended and undulate.

Intra-thoracic tumors and cysts as far as physical signs are concerned frequently very closely resemble pleuritic effusion. They may displace the heart, compress the lung and cause disappearance of the respiratory murmur with percussion dulness. The points of difference are as follows: The area of dulness is usually situated in the upper and middle portions of the chest, it is more circumscribed than in pleurisy, while fremitus is often preserved as in consolidation. The history of the case is also an important diagnostic factor as it necessarily materially differs from that of pleurisy. Pleuritic effusion may sometimes be associated with intra-thoracic growths, especially when malignant. The diagnosis under these conditions may be assisted by exploratory puncture or it may be impossible until pressure symptoms and cachexia develop.

Hepatic abscess or cyst when large and suitably located may displace the diaphragm pushing it upward and cause dulness and diminution or absence of respiration at the lower portion of the right pleura, and thus present a strong resemblance to a pleuritic effusion. In such cases the upper line of dulness is convex, and a friction murmur is audible over the area of dulness which could not occur if effusion were present.

Additional difficulties in diagnosis may appear in certain cases of extensive pneumonic infiltration, where instead of the usual percussion dulness there is absolute flatness, and weakness or absence of the respiratory murmur instead of bronchial breathing, absence of râles, expectoration and increase of fremitus. In these cases there may also be an insidious onset and absence of initial chill. Again in children small exudates may be impossible to recognize by the physical signs. In both these conditions the diagnosis rests upon the result of exploratory puncture.

Prognosis.—The outlook in sero-fibrinous pleurisy is governed chiefly by the resistive force and vitality of the patient. As a rule it is always difficult to form any definite or positive opinion as to the future outcome in a given case before the beginning of the fourth week.

Small effusions generally disappear rapidly, while large may require weeks for absorbtion. In cases with tuberculous tendencies the diminution of the fluid is gradually very tedious, or it may persist for many months and resist all treatment. Delay of absorbtion beyond the third week must be regarded as unfavorable since it indicates that the inflammatory process is great and the vitality of the patient is impaired, with the result that the tissues of the pleura are probably permanently weakened.

Refilling of the pleural cavity at the end of about a week after evacuation must be looked upon as of rather unfavorable significance, refilling after a second evacuation as more unfavorable.

Bacteriological examination of the exudate has an important prognostic bearing. The presence of pneumococci has a favorable significance (this is denied by some German writers). Cases of this type generally recover. The presence of, on the other hand, of streptococci is of unfavorable significance, as it points to purulent infection, but only in rare instances are these micro-organisms found without pus, especially the streptococci. A sterile effusion suggests tuberculosis. In the varieties called secondary the prognosis depends upon the primary cause, which frequently overshadows the pleurisy.

Double pleurisies are very unfavorable except those of rheumatic origin and those occurring during the puerperium when recovery may take place. Cases which develop sweats, high continued fever or sweats with mild fever should be examined for pus.

After resolution in many cases there may remain in the apex of the affected lung slight dulness or loss of the respiratory murmur. This does not necessarily signify that there is a commencing tuberculosis, for in many instances it is simply due to imperfect expansion of the lung and is not unfavorable. Again the lung may never fully regain its tone. This lack of tone may persist or adhesions may form and become permanent and ultimately chronic bronchitis and emphysema develop.

In some instances again after the acute symptoms have subsided convalescence runs a tedious course, the physical signs remain for many weeks and the patient fails to fully recover his strength. These are the cases which are prone to ultimately develop pulmonary tuberculosis. Those which thus terminate are undoubtedly tuberculous from the onset.

Sudden death is very rare, but may occur from œdema of the lung on the unaffected side, degeneration of the myocardium, embolism and thrombosis of the pulmonary artery or of the heart itself, or mechanical impediment of the circulation from displacement of the heart (Weil).

Special Forms of Acute Pleurisy.

Hæmorrhagic Pleurisy.—This variety is that in which the serous effusion is mixed with blood. It may occur in the following conditions: Tubercular infections following tuberculosis of the lung, cachexias and depraved conditions of the constitution, such as alcoholism, scurvy, purpura and debility of age, malignant disease, cirrhosis of the liver, Bright's disease and acute infections. In some cases it is difficult to assign any definite cause.

There are no symptoms or signs which positively denote a hæmorrhagic condition of the effusion in pleurisy. Sudden pallor has been mentioned as denoting the sudden occurrence of hæmorrhage, but this symptom may appear with many other conditions. In sero-fibrinous pleurisy its appearance in association with any of the above casual conditions may be regarded as suggestive. Exploratory puncture, however, is the only positive means of determining the condition. It is important to observe that a hæmorrhage may be accidentally caused by puncture of the lung in aspiration.

In hæmorrhagic serous-pleurisy the outlook depends upon the cause. Obviously from its ætiology in many instances this is unfavorable, although the presence of a moderate amount of blood does not in itself otherwise necessarily lend an especially serious aspect to the case.

Encapsulated or Encysted Pleurisy.—Mention has been made of the pathological changes which result in the formation of pouches or cavities by the presence of adhesions between the costal and pulmonary pleura. These cavities vary in number and extent and may be located in any part of the pleura. They are rather more frequent in empyema than in serous effusions. Diagnosis is very difficult. The absence of tactile fremitus and the presence of dulness along certain lines and in certain areas of the chest may be regarded as very suggestive, especially so when present with the general symptoms of pleurisy. Repeated exploratory punctures, however, are necessary for diagnosis.

Interlobar Pleurisy.—This is the accumulation of the effusion between the lobes of the lung. It is not difficult to comprehend how a pleuritic inflammation might in extending over the surface of the pulmonary pleura involve that portion between the lobes, but the term is rather applied to the inflammatory process, when it results in adhesions of the surfaces of two adjacent lobes of the lung and thereby forms a pocket or sac containing the products of inflammation. This condition is more frequent on the right side and at the root of the lung between the upper and middle lobes. It is to all intents practically a cyst containing a serous or purulent effusion. In the latter instance it may simulate pulmonary abscess and form a fistula opening to a bronchus, but such a condition is rare.

The cause is not always certain. In some instances it follows croupous pneumonia, in others it may result from a focus of tubercular infection, in others again it has been found to be associated with an old pleurisy. Pneumothorax is a rare accident in the purulent form.

The symptoms are indefinite in the purulent form. They resemble pulmonary abscess, being hectic, chills and a circumscribed area of dulness surrounded by resonant tissue. There may be the sudden appearance of putrid purulent expectoration.

It is not possible in life to differentiate [illegible] purulent pleurisy and abscess of the lung. The [illegible] favorable. Some advise, therefore, [illegible] others advocate removal by aspiration [illegible]

Diaphragmatic Pleurisy.—The [illegible] flammatory process is confined to the diaphragmatic pleura or involves it in conjunction with [illegible] portions of the pleural surface. The [illegible] sero-fibrinous or purulent. [illegible] instance of serous effusion the [illegible]

The symptoms consist of [illegible] referred to the epigastrium [illegible] rib to the xiphoid cartilage [illegible] ognomonic. The [illegible] swallowing, when [illegible]

is a pronounced symptom. Sometimes the patient is
sume a sitting posture or bends forward. The respira-
ck, superficial and purely thoracic. The abdominal
fixed, cough, nausea and vomiting may be present. A
e condition is that the dyspnœa may frequently be ex-
ccount of the pain even when the inflammatory process
that a pronounced and apparently dangerous degree
may develop. The subsequent course, however, usu-
ows that the danger is more apparent than real. The con-
onal symptoms are generally marked, more especially the
fever, which sometimes is higher than in the ordinary forms of
pleurisy.

The physical signs are often negative, in fact, the presence of subjective symptoms without physical signs is characteristic of the disease.

If the effusion is purulent there may be bulging of the lower intercostal spaces.

Tuberculosis of the Pleura.—In view of the fact that tubercular infection enters so largely into the ætiology of all forms of pleurisy, it seems scarcely tenable to regard tuberculous pleurisies as special types of the disease, but it may not be amiss to discuss the question solely from the standpoint of tubercular infection.

Infection of the pleura by the bacillus tuberculosus occurs primarily and secondarily.

I. Primary infection causes the following:

(1) Acute fibrinous and sero-fibrinous pleurisy. The course of these processes is as discussed. Cases which after the acute stage run a chronic course and those of the subacute and chronic type are commonly tuberculous as elsewhere mentioned. (2) Acute suppurative pleurisy: Some of the acute purulent forms of pleurisy are distinctly tuberculous, at the same time the infection is mixed. These pleurisies commence with sharp symptoms, fever, cough, pain and frequently chill. There may be no external evidences suggesting tuberculosis. *Vide* empyema.

(3) Chronic adhesive pleurisy. See that subject.

II. Secondary infections.—These are very frequent and are essentially part of the pathological change in tuberculosis of the lungs. The process is usually chronic, but acute tubercular pleurisy may occur in the course of a pulmonary process, with a serous, hæmorrhagic or purulent effusion. A condition possible under these circumstances is perforation of the lung into a bronchus and the formation of pyo-pneumothorax.

III. Latent pleurisy.—This form mentioned by some writers as a variety of the acute type is considered in the discussion of chronic pleurisy.

Treatment.—The general measures as to rest in bed and diet should be followed as discussed in the treatment of the fibrinous form of the disease. It is desirable to enjoin as dry a diet as possible. The special indications for treatment are limitation of the inflammatory process, absorbtion of the effusion and support of the patient's strength by simple nutritious food.

First Stage.—During this period the local measure and internal special medication as mentioned in the fibrinous variety are indicated for the relief of pain and limitation of the inflammatory process.

Second Stage.—The importance of dry diet needs here to be emphasized. The following remedies are of service in promoting absorbtion.

Bryonia is indicated by the presence of sticking pain in the side, but is of limited utility after effusion has taken place, being ineffective when it is excessive.

Cantharis is much lauded by the French. It is generally regarded as one of the most important in sero-fibrinous exudation. Ten drops of the tincture in four ounces of water, a teaspoonful every three hours, constitute an effective method of administration. Jousset gives the third decimal of dilution, saying that if this fails, give the tincture.

Apis mel., after cantharis the most important remedy for the removal of the effusion.

Arsenicum album may prove of service after the failure of cantharis and apis if there is increase of the effusion with cyanosis and dyspnœa, restlessness and anxiety.

Colchicine, when gout or rheumatism is the cause, with perspiration which smells sour and scanty, turbid and albuminous urine.

Kali iodidum sometimes will prove beneficial and should be tried when other remedies fail. About ten grains three times a day is a suitable dose.

Sulphur, when the general symptoms point to this remedy, together with want of reaction.

Hydrogogue cathartics and diuretics are generally without effect. Free diaphoresis has been employed and sometimes assists, but it is dangerous if the heart is at all weak. The salicylates have been praised by some, but are generally inferior to the remedies mentioned. Iodine locally is used, but it is doubtful whether it has much effect.

Thoracentesis.—In a certain proportion of cases, about one-third, all attempts to promote absorbtion of the effusion fail. Recourse must then be had to thoracentesis. The indications for this measure may be viewed from two standpoints, namely, imminent danger to life which may arise during the febrile stage and persistence of the effusion without immediate urgency of symptoms, as it occurs after the subsidence of fever.

(1) The presence of the following symptoms demands immediate thoracentesis: excessive orthopnœa; syncope with cyanosis, pronounced displacement of the heart, especially when murmurs develop, copious effusion with signs of involvement of the right side as evidenced by râles, bronchial breathing and impaired resonance, rapid filling of the pleural cavity, complete filling of the cavity and double pleurisies.

(2) In the conditions which call for thoracentesis apart from emergencies the chief aim is the prevention of permanent change in the lungs. Under these circumstances the indications for the measure are absence of any evidence of diminution of the effusion after the temperature has been normal a week and subacute conditions when the temperature has been normal or nearly so from the onset with persistence of the effusion. When these indications are present, thoracentesis should not be deferred for more than a week. In general it may be said that delayed resolution is an indication for thoracentesis. Another general indication is that when the effusion reaches the clavicle no matter how free from discomfort the patient may be, the chest should be aspirated.

The presence of fever is not a contra-indication. Sometimes the fever will fall after aspiration.

Thoracentesis is contra-indicated by the presence of croupous pneumonia, marked debility, the feebleness of old age, and is usually uncalled for in children.

The procedure will prove in many instances more than merely palliative as in some cases it is followed by resolution.

The instrument used should be Dieulefoy's aspirator. In absence of this, a simple fountain syringe may be connected with a trocar (all sterilized). The syringe is filled with an antiseptic solution and is immersed in a vessel containing the same. Puncture is made and the effusion flows out behind the antiseptic solution.

Thoracentesis if properly performed is free from danger. The following is its technique. The patient rests in bed in a semi-recumbent position, the ribs should be separated by bringing the arm of the affected side around across the chest with the hand on the shoulder of the well side. The point of puncture is the sixth or seventh interspace on the right side, and the fifth, sixth or seventh interspace on the left—both in the mid-axillary line. Sometimes puncture may be made in the back, especially when there are adhesions which prevent the effusion from extending far beyond the posterior axillary line. Under these conditions the operation should be performed with the patient in the sitting posture in order to permit of the better expansion of the lungs. When puncture is made posteriorly some surgeons select a point just below the scapula or one in the seventh, eighth or ninth interspace in the posterior axillary line. The surface of the skin should be prepared by scrubbing with green soap, then bathed with ether, then with bichloride solution and covered with bichloride gauze. The assistant should draw up the skin from the interspace and the operator should use the forefinger as a guide. The hands of both should be rendered aseptic. The needle should be inserted quickly, close to the margin of the rib on the lower border of the interspace, that is the inferior rib, care being taken not to wound the periosteum.

The amount of fluid drawn at one time will depend upon the extent of the effusion. If the fluid reaches to the clavicle a pint may be removed safely. Usually never more than twenty-four ounces should be withdrawn except in emergencies in the febrile stage. Under these conditions the lung being subjected to a short period of compression permits of a more complete removal of the fluid exudate. The fluid should be allowed to flow slowly so as to permit the lung to expand gradually. This care is most essential, as too rapid expansion of the lung is liable to be attended with sudden pulmonary congestion and œdema which may quickly terminate fatally.

If, during the withdrawal of the fluid, cough, faintness, dyspnœa and constriction around the chest appear, the needle should immediately be withdrawn. During the flow of the fluid the needle should be held by the operator in order not to cause the patient pain. It should be drawn out slowly; after its removal the puncture should be covered with an antiseptic plaster or collodion and dressed with antiseptic gauze. Some oozing is frequent, but it is without special significance. In some instances, notwithstanding the physical signs of the presence of fluid, thoracentesis may prove futile for the reasons mentioned in the discussion of the diagnosis.

Certain symptoms and accidents which may possibly occur during thoracentesis: Some are of common occurrence while others are rare. The former are the following: pain of a sharp, knife-like character after the withdrawal of a certain amount of fluid, coughing towards the end of the procedure and faintness. Rare accidents are: subcutaneous emphysema at the point of puncture without pneumothorax, pneumothorax, albuminous expectoration, a condition characterized by dyspnœa and usually fatal, epileptiform convulsions and sudden death from syncope or during a convulsive seizure.

During convalescence tonics are generally indicated. The food should now be of a more substantial nature and of the highest nutritive qualities. The patient should spend a good portion of the time in the open air. In fact, the general management of the after consequences and convalescence of sero-fibrinous pleurisy is much on the same line as that of pulmonary tuberculosis.

Breathing exercises should be practiced daily as mentioned in the treatment of empyema.

3. Empyema.

SYNONYMS.—*Purulent Pleurisy.*

Empyema is inflammation of the pleura accompanied by purulent effusion.

Ætioloy.—Bearing in mind that all types of pleurisy, generally speaking, depend upon the same causal influences, the special ætiology of empyema is limited to the consideration of the conditions under which these factors may result in purulency of the inflammatory process.

Empyema always arises from infection by micro-organisms. In the discussion of the bacteriology of pleurisy it will be recalled that infection of the tubercle bacillus and the micrococcus lanceolatus may be followed by either serous or purulent effusion, while infection by the streptococcus pyogenes aureus commonly results in purulency, and is usually associated with sepsis. Many other micro-organisms are found, among the most frequent are the staphylococcus pyogenes aureus which is common in pleurisies associated with suppuration in other parts of the body, and the leptothrix pulmonalis in putrid effusion. A sterile exudate is suggestive of tuberculosis. Purulent infection by the pneumococci, as a rule, runs a comparatively favorable course, while those due to the streptococci necessarily are the most unfavorable.

Empyema may arise as a sequel of sero-fibrinous pleurisy or the effusion may be purulent from the beginning. Thus there are two types of development, viz. :

(1) In this variety it is difficult in many cases to find a satisfactory explanation for the change except that it be attributed to debility or dyscraisa. Usually in healthy subjects a serous-effusion remains serous. In children the effusion may rapidly become purulent early in the disease. Again purulent infection may follow thoracentesis.

(2) The second variety occurs with the following affections :

(*a*) Acute infections, namely, scarlet fever, pneumonia, broncho-pneumonia, septico-pyemic diseases, tuberculosis and dysentery. These are the most frequent causes, among which scarlet fever stands first; it is well known that pleurisies arising with scarlet fever are generally purulent. Less frequent causes are erysipelas, typhoid and pertussis. Typhoid pleurisies are most always purulent. (*b*) Chronic tuberculosis and tuberculous cavities which perforate the pleura. (*c*) Gangrene, abscess, hydatids, actinomycosis of the lungs. (*d*) Malignant disease of the lungs and adjacent structure. (*e*) Purulent pericarditis, abscess of the mediastinum. (*f*) Peritonitis, subphrenic abscess. (*g*) Injuries to the chest walls. (*h*) Caries of the ribs or vertebræ.

Morbid Anatomy.—On post-mortem examination of the pleural cavity in empyema there is found either a sero-purulent effusion separated into layers, a greenish-yellow layer above with a thick cream-like layer below, or simply a turbid effusion with floating flocculent matter, or a thick creamy pus. The first is most frequent. The odor of the purulent exudate is usually sweetish, but under certain conditions may be putrid.

Putrid effusion is more frequently associated with thoracic or pulmonary fistula, but may occur in the absence of either. It also is found in association with traumatism, pulmonary gangrene and abscess where the odor is very foul. In the pleuritis acutissima of Fränkel the effusion is liable to be of that nature.

Putrid effusion may tend to extensive necrotic processes, ulcers, perforations, erosions and abscess.

Microscopical examination shows the exudate to be the same as that observed in purulent inflammatory processes elsewhere.

The pleura is greatly thickened, sometimes to the extent of 1 to 2 mm. The changed membrane is made up of newly formed connective tissue, new blood-vessels and leucocytes. The surface is grayish-white with granulations which after the disappearance of the pus result in the formation of firm adhesions.

The costal pleura may show the presence of erosions and both it and the visceral pleura may undergo perforation, especially in cases of long standing. The lung is much compressed. In some instances the pus may become encapsulated between the liver and diaphragm either due to direct rupture or to extension of the inflammatory process. This condition is uncommon.

In exceptional instances the pus may be absorbed or undergo inspissation, becoming a thick layer of the consistency of butter, or it may undergo a more or less cheesy degeneration and eventually become calcareous. The last change is very rare.

Spontaneous discharge may sometimes occur, taking place most frequently through the lung or bronchi, more rarely through the chest wall anteriorly beneath the ribs. In the latter instance a fistulous opening remains with pneumothorax constituting a condition of pyo-pneumothorax.

Empyema is almost always unilateral. The disease has occurred, however, on both sides.

Symptoms.—As foreshadowed in the discussion of the ætiology the symptoms attending the formation and presence of pus in the pleural cavity are variable. There are frequently evidences of sepsis, chills, fever, sweats and prostration. The chills are diurnal and irregular, the fever also irregular and the sweats pronounced. There is pain in the side which is intensified by breathing, movement, cough and dyspnœa. Peptonuria is present, and while in no way pathognomonic, as it occurs in other suppurative processes, when taken in connection with other symptoms, is of diagnostic significance, inasmuch as it serves to eliminate sero-fibrinous pleurisy. The urine also shows the presence of indican and the blood decided leucocytosis. While these symptoms may be said in a general way to typify empyema the variations in its clinical manifestations range from symptoms of an acute septic nature to conditions of latency.

The onset may be either sharp and sudden, followed by acute symptoms, or gradual followed by those of less intensity, or insidious when the condition remains more or less latent. Most frequently empyema follows a sero-fibrinous pleurisy or comes on insidiously. The disease has been divided by some writers into the acute, subacute and chronic varieties.

Acute empyema may be primary and purulent from the beginning. There is an initial chill, pain in the side, cough and dyspnœa. The fever varies in severity, yet there may not be anything pointing definitely to the presence of pus. A form of acute pleurisy is that described by Fränkel (pleuritis acutissima) in which there are very acute symptoms, pain in the side, rigors, high fever and the pus develops with great rapidity. There are marked symptoms of sepsis with rapid emaciation and the patient sinks into the typhoid state and dies. The course of this unusual form of empyema may not be more than one week.

The subacute variety is much more frequent. There is pain in the chest of a mild character, some shortness of breath and mild fever. In many instances the symptoms appear to be those of sero-fibrinous pleurisy with nothing at first to suggest the presence of pus, but later the temperature curve arouses suspicion that the effusion is purulent. In these cases the temperature instead of falling to nearly normal at the end of a fortnight persists and shows an evening elevation of 102° to 103° F. Soon hectic develops and the symptoms of sepsis become more apparent, there being chills or chilly sensations, exacerbations of fever, pallor, sometimes a slight puffiness of the face, occasionally night-sweats, loss of appetite and progressive emaciation.

Chronic empyema may be entirely latent. Fever may be mild or absent and sometimes there is an occasional subnormal temperature. The patient may be quite unaware that there is any disease of the thorax. There is, however, general loss of health and malaise.

In view of the invariably infectious nature of empyema its consideration according to the variety of the infecting micro-organism is advisable.

Pneumococcus empyema may run either an acute, subacute or chronic course. Cases of sero-fibrinous pleurisy which subsequently become purulent are often of this type of infection.

According to Netter about 60 per cent. of pneumococcus empyemas are attended with fever, pain in the side and dyspnœa. In some there is an initial chill, cough and even herpes, not unlike pneumonia. In others the fever is continuous and chills are rare. In others again the development may be quite indefinite. (Compare meta-pneumonic empyema.) Pneumococcus infection may also assume the latent type and develop without pain or constitutional symptoms. Spontaneous evacuation is rather more frequently associated with it. It is rarely attended with œdema of the affected side. The pus is rich in cells, very viscid, and somewhat greenish in color. These features, however, may be absent. The comparatively favorable course of pneumococcus empyema is noted in the prognosis.

Empyema as a sequel of pneumonia generally appears in the second month when the symptoms of the pneumonic process have subsided.

Streptococcus empyema, while characterized by a degree of diversity in its manifestations, is more closely associated with acute symptoms of sepsis than either of the other forms of infection. Many of the cases which appear purulent from the beginning are of this type. It also includes those varieties of empyema which are acute in their onset and course, and which are characterized by chills, fever with deeply oscillating temperature curve, profound prostration, rapid emaciation and sometimes a rapidly developing typhoid state. Again under this form of infection are found types of empyema in which the symptoms are of a more moderate degree of intensity; those cases which begin as sero-fibrinous pleurisy and subsequently become purulent, and those in which the fever is mild but continuous. Finally the onset and course of the infection may be latent.

The pus of the streptococcus empyema is of a yellowish color and separates on standing into an upper or serous layer, which is opaque, and a purulent layer below. It is important to note that this separation may likewise occur in the chest cavity.

Tuberculosis bacillus empyema is characterized by insidiousness of onset and chronicity of course. It is frequently secondary and may be purulent from the beginning. It may occur with tuberculosis or infection may take place during the convalescence of an attack of pneumonia. The symptoms are ill-defined. There is a general decline of health, malaise and possibly slight, dry hacking cough. Fever is slight, absent, or the temperature is subnormal. In very exceptional instances the onset of tuberculosis empyema may be abrupt, and the course rapid, usually terminating in death. The pus is thin, sero-purulent and grayish in color. On standing it precipitates a yellow deposit.

While the nature of the infection is very important it will be observed that although certain types of the disease predominate with the certain varieties of micro-organisms there is no constant relationship.

The course of empyema with the exception of the rare acute varieties mentioned is decidedly chronic. Small effusions, especially if due to the pneumococcus, may be absorbed in rare instances, less frequently in tuberculous infection and almost never in those of the streptococcus.

PHYSICAL SIGNS.—The physical signs of purulent pleurisy are with certain modifications identical with those of the sero-fibrinous variety. *Inspection* shows bulging of the affected side, especially in children, when the disproportion between the two sides is sometimes excessive; the intercostal spaces may even bulge. Distention of the veins of the neck and œdema of the chest which is rare in serous effusions, is more frequent. In empyema necessitatis a protrusion at the point of prospective rupture with reddening of the surface is usual. In cases of spontaneous rupture the opening will be observable. Sometimes there are several fistulæ. *Palpation* is practically the same as in sero-fibrinous pleurisy. *Percussion* shows that there is more displacement in a given amount of effusion when the latter is purulent than when it is serous. This is probably owing to the greater weight of the pus. *Auscultation* is the same as in serous-pleurisy. Baccelli's sign is mentioned by many writers, viz.: That the transmission of whispered resonance over the area of flatness occurs only when the effusion is serous is without value, for, as stated elsewhere, large serous effusions sometimes fail to transmit the whispered resonance and purulent may transmit resonance. In children the respiratory sound may be loud and tubular over effusion of pus of considerable size.

The physical signs of pulsating pleurisy do not call for separate consideration, they are simply those of pleurisy with pulsation added. The pulsation is synchronous with the heart and with rare exceptions is on the left side.

SPECIAL FORMS OF EMPYEMA.

Empyema Necessitatis.—In addition to the points of rupture aforementioned discharge of pus in empyema may take plàce through the chest walls constituting the condition called empyema necessitatis. This is rather favorable. The perforation of the chest wall may take place at any point from the third to the sixth rib, usually in the fifth interspace anteriorly. It may also occur posteriorly at the angle of the scapula. Penetration may occur at more than one point and the internal and external opening may not be in apposition, but may be some distance apart, being connected by a fistula. Attending the development of perforation of the chest wall there is peripheral inflammation, œdema, heat, pain and finally fluctuation.

Meta-pneumonic Empyema.—Netter mentions this as a special variety. In occurs in children and in adults up to about the fortieth year. It generally, though not invariably, follows pneumonia which has not terminated by crisis. It is frequently epidemic and is characterized by rise of temperature, immediately following a brief afebrile period. The purulent effusion is generally thick with much fibrin and numerous pus corpuscles. Frequently it is encapsulated. The fever is continuous. The prognosis in these cases is claimed to be rather more favorable than in other types. The cause of the process in uncomplicated cases is the pneumococcus.

Encapsulated Empyema.—The pus may become encapsulated in the pleural cavity as in the serous variety, the pathological process differing in the nature of the effusion. There may also be a primary encapsulated purulent pleurisy.

Interlobar Empyema.—The empyemic process may also take this form, the seat of the purulent process being located between the lobes of the lungs. The fact should not be overlooked that serous, purulent, hæmorrhagic and putrid effusions may exist in various cavities in multilocular pleurisies. Compare Interlobar Pleurisy.

Pulsating Empyema.—This is a phenomenon of comparative variety. It consists of a periodic pulsation of the entire half or a circumscribed area of the thorax. The condition only occurs in purulent exudates and usually with empyema necessitatis, although not always so. The pulsation is almost always systolic and confined to an area of two or three inches, although it may be widely dispersed as first mentioned. With rare exceptions it occurs on the side of the chest, on its antero-lateral aspect. Posterior pulsation is rare. The cause of pulsation in empyema has not yet been satisfactorily explained. From the fact that the pulsation ceases when even a small quantity of the pus is removed suggests, according to Bouveret, that the cause of the phenomenon is a certain degree of tension of the chest walls.

Other explanations which which have been offered are the presence of a copious effusion, paralysis of the intercostal muscles with consequent relaxation of the chest walls and forcible beat of the heart.

Peripleuritis.—This is inflammation and suppuration of the layer of connective tissue between the costal pleura and muscular layer of the chest wall. Practically it cannot be clinically separated or distinguished during life from ordinary pleuritic inflammatory processes.

Diagnosis.—If the symptoms of hectic and sepsis are present with the signs of pleurisy with effusion, the condition may be inferred to be empyema. But in this, as well as in other instances when the symptoms are indefinite or negative, an absolute diagnosis rests upon the result of exploratory puncture. If the first puncture is negative it should be repeated, especially if there are typical symptoms present, such as fever and sweats.

The most important feature in the differentiation of empyema is to distinguish it from sero-fibrinous pleurisy. Those cases in which the fever runs high and the symptoms of hectic or sepsis are in evidence are usually easily determined, but in the many instances when the symptoms are less positive and the fever mild, the diagnosis is difficult. Under these conditions exploratory puncture should be made. An important feature in empyema is the presence of leucocytosis and peptonuria, both of which are absent in sero-fibrinous pleurisy. Again, inasmuch as empyema in some of its types may be frequently confused with those conditions which simulate sero-fibrinous pleurisy, it follows that what have been mentioned in regard to the differential diagnosis of that affection, applies with some modifications to that of empyema.

Pulsating empyema must be differentiated from aneurysm of the thoracic aorta. It may be distinguished by the following features: empyema is often on the left side, it is some distance from the median line, it is attended with quite extensive percussion dulness at the base and posterior portion of the chest, and murmurs synchronous with the heart are absent. For further points of difference the reader is referred to page 382.

Abscess of the chest wall and peripleuritic abscess (peripleuritis) are extremely difficult in some instances to distinguish from empyema necessitatis. If the abscess is circumscribed and presents a well-formed tumor with the area of dulness limited to the same, the diagnosis can usually be made, but if the dulness extends beyond there are no positive points of distinction. Additional difficulties present, if the abscess is complicated with effusion or if there is œdema of the chest walls which impairs the percussion-note. Dulness in empyema is, however, generally much more regular in its conformation or outline than in infiltration of the walls of the thorax.

In all cases of empyema examination of the pus should be made and the nature of the infecting micro-organisms carefully determined.

Prognosis.—Empyema in all instances is a grave affection. If left to itself with few exceptions it ends in death. The presence of the prolonged suppurative process saps the vitality or leads to amyloid degeneration of the kidneys and other viscera, and the patient dies of asthenia. Many cases succumb to tuberculosis. The most favorable outlook is in pneumococcus infection. The limited number of cases which resolve by absorbtion are generally of this nature.

Among children the prognosis is more hopeful. Rupture externally may be regarded as fortunate. Rupture into the bronchial tubes may sometimes also be regarded as favorable, although it may result in sudden death by inundating the lungs. The prognosis may be rendered much more favorable by prompt surgical treatment. In fact the outlook depends upon the results of operative interference.

In favorable cases there will always remain more or less obliteration of the pleural cavity, impairment of the functions of the lung with retraction of the affected side. In bilateral empyema the prognosis is naturally more grave.

On the other hand, unless there is surgical interference the pus may not only burrow and break into the lung, discharge through the bronchi and give rise to pneumothorax, but it may also, according to Traube, cause necrosis of the pulmonary pleura and escape by soaking through the spongy tissues of the lung without the formation of pneumothorax. It may likewise penetrate neighboring structures such as the œsophagus, pericardium, mediastinum, stomach or peritoneum. In exceptional instances it may burrow along the vertebral column, penetrate the psoas muscle and eventually appear in the iliac fossa where it may simulate psoas abscess.

Treatment.—The management of empyema is on strictly surgical lines. It consists of the prompt evacuation of the pus and the establishment of free drainage. It has been asserted by certain clinicians that cases operated upon after the first month do better than those that are operated earlier. This view, however, has not been generally accepted and the safest rule is to evacuate the pus as soon as its presence is recognized, for delay is dangerous. The several methods of treatment are thoracentesis, siphon drainage, simple resection of a rib and thoracoplasty.

Thoracentesis.—This is limited in its application. Occasionally in children when the infection is due to the pneumococcus it may be followed by recovery, but too much reliance should not be placed upon it. If aspiration has been performed once or twice, and if the temperature does not fall within a week, an incision should be made. Aspiration may prove of service in conditions where the symptoms are very urgent, such as pulmonary œdema, excessive dyspnœa and extreme exhaustion. It must not, however, under these circumstances be regarded as a substitute for incision or resection, but only as a temporary expediency, when it should be followed by either of the other measures.

In performing thoracentesis for purulent pleurisy the same general rules of asepsis and antisepsis as laid down for thoracentesis in the instance of sero-fibrinous pleurisy should be rigorously followed. The same precaution should also be exercised. Alcoholic stimulants should be given before the operation to anticipate and prevent syncope. If the pulse becomes feeble during its procedure place the patient upon his back; if a cough or a sensation of fainting appears, discontinue.

During the operation the patient should be supported by pillows in a semi-recumbent position with the arms raised over the head.

Anæsthesia is not necessary. The skin may be rendered anæsthetic by the chloride of ethyl or ether spray, or a drop of pure carbolic acid. The point of puncture will be according to circumstances, if the dulness on percussion is more marked at any one point, or if there is bulging, these localities may be selected, otherwise the needle may be inserted in the midaxillary line in the fifth intercostal space on the right side in front of the latissimus dorsi muscle or in the sixth space on the left. The puncture should be made just above the upper border of the lower rib of the selected interspace.

Incision.—This procedure is proper in empyema of children and in recent cases in adults when the intercostal spaces are sufficiently wide to admit of free drainage. In cases of long standing there is often some retraction of the chest wall with consequent narrowing of the intercostal spaces; under these conditions incision is inadequate. The technique is as follows: Render the skin thoroughly aseptic by cleansing with green soap, alcohol or ether and bichloride, according to the usual methods. Next anæsthetize the surface with ethyl chloride unless general anæsthesia is employed, in which case chloroform should be preferred. The degree of narcosis should be moderate, as the operation should be completed inside of three minutes. Place the patient in the lateral semi-recumbent position with the arms raised above the head and lower him as the evacuation progresses unless there is empyema necessitatis. The point of incision should be in the fourth or fifth interspace or under special conditions in the sixth. The incision should begin at the anterior axillary line and extend at least two inches laterally outward; it should be sufficiently large to admit of the introduction of two fingers which should be inserted as soon as the opening is made in order to prevent the too rapid evacuation of the pus and to break up the false membrane and thus facilitate its removal. This procedure is said to greatly enhance the healing process.

In making the incision care should be exercised to avoid wounding large veins. The skin, the first layer of superficial fibres and the intercostal muscles, should be divided layer by layer until the pleura is reached, then this should be cut through with the bistoury; the wound should now be lengthened either to the right or left. After the evacuation of the pus insert two drainage-tubes about eight or ten inches long. Care must be exercised that their inner extremities do not impinge upon the lung, thoracic wall or diaphragm. The use of two tubes is advised in order to facilitate the flow of the pus and to permit of free drainage in case one tube becomes clogged. The introduction of the tubes should be facilitated by the application of vaseline, after which the greased ends may be dusted with iodoform. The tubes should be held in position by a large safety pin passed through their outer extremities. A packing of soft cotton should be used to prevent the pin from pressing upon the soft parts and the whole dusted with iodoform. Simple iodoform dressings should be used.

Siphon Drainage.—This method was suggested by Bulau. It consists of puncture under strict antisepsis combined with permanent aspiration. Its application is as follows: The puncture is made as in ordinary thoracentesis, at the same time a Nelaton catheter is introduced through the cannula; after the removal of the latter the catheter is secured in position by means of collodion and cotton dressings. A long rubber tube into which a glass tubing is adjusted is now connected with the cannula and its distal extremity is immersed in a vessel containing an antiseptic fluid. The effect is that of a siphon which gradually draws off the effusion and allows the lungs to slowly expand. Bulau's method it has been remarked has been endorsed by clinicians rather more favorably than by surgeons. It is certainly conservative. Disadvantages are the necessity of the patient remaining upon his back for a considerable length of time and the possibility of dislodgment of the tube and its occlusion by shreds and tissue detritus. The latter accident may be avoided by the use of a T-shaped piece of glass tubing as suggested by Fränkel. This is attached to the drainage-tube. A stop-cock is placed in the main tube near the thorax, if the tube becomes clogged, cut off the main tube and force the fluid through the other so that it will come out of the occluded tube and clear away the detritus.

Revilloid used a method similar to Bulau, differing in that there was a rubber bulb about two feet from the proximal extremity of the rubber tube. He recommended this for intractable cases which resisted the classical methods of treatment.*

Resection.—The majority of clinicians favor this procedure for adults when anæsthesia is allowable; in fact it is the most preferable method. The operation is indicated even when the pus has evacuated through the lungs or burrowed through the chest walls. There are those, however, who still maintain that, even when contraction of the intercostal spaces is so extreme that the little finger can be barely introduced and the introduction of a medium-sized drainage-tube cannot be accomplished without compressing it, the opening may be enlarged by dilatation in a few days sufficiently to admit the index finger, and a resection thus rendered unnecessary. Nevertheless the best results have been obtained by resection.

When it is decided to perform resection, chloroform anæsthesia is generally preferable. Prepare the patient as regards asepsis and antisepsis as usual. It is advised that after unconsciousness the patient be not turned. The incision should be made over the middle line of the selected rib, usually the sixth, to the length of about three inches, exposing the rib and cutting through the periosteum. The edges of the wound should be held apart with retractors. The periosteum is next bisected by a horizontal incision the width of the rib, then raised with the surrounding tissues and pushed to each side. The intercostal artery may now be separated with the periosteum; if cut it must be ligated. The denuded rib should next be seized with the forceps and cut either with the bone forceps or rib cutter, removing about one and a half inches. The pleura is now to be opened by a small incision and the pus allowed to drain slowly. Insert the finger and sweep it in all directions within the cavity to clear away any obstructing adhesions. The finger also acts in regulating the flow of the pus. When the patient has recovered consciousness to a certain extent, his position may be changed so as to facilitate the flow of pus. After evacuation insert a drainage tube the size of the little finger and secure it with a safety pin, sew up the edges of the wound around it, dust with iodoform and dress with the same, covering the whole with a pad of sterilized cotton, securing with bandages. The dressing should be changed when saturated or when the temperature rises. Usually a change will be necessary every second or third day. The tube should be shortened at each dressing according as the process of healing progresses. The condition of the cavity may be ascertained by careful probing with a soft rubber catheter.

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* Digest of External Therapeutics, E. G. Rankin, M.D., 3d Ed., New York, 1904. Treatment of So-called Incurable Purulent Pleurisies by the Siphon. C. G. Cunningham, M.D., Boston Medical and Surgical Journal, November 22, 1904.

Posture may be called into requisition to assist in drainage. Place the patient on the affected side, raise the hips, lower the shoulders, then reverse the movement, repeat a few times. This may be done several times daily. For further details of resection see works on surgery.

Thoracoplasty.—This operation consists of the resection of several ribs. (Estlander's operation.) It is one of gravity and is indicated only in cases of long standing which have resisted other methods and where a fistulous opening persists. Schede has modified the operation, when expansile power of the lung was lost and the walls of the pleural cavity were unable to adjust themselves to the lung on account of their rigidity, by closing the thoracic walls. He removed not only the ribs, but the rigid thoracic walls (intercostal spaces) over the entire extent of the empyemic cavity. A flap consisting of skin, muscle and scapula is made, which adheres and adapts itself to the lung. Deformity at first is marked, but in time becomes much modified. It is claimed that the thorax eventually almost regains its normal appearance.

Schede succeeded in curing five cases out of seven. A favorable result when it is considered that almost all cases of this nature have succumbed to either amyloid degeneration or asthenia.

Irrigation.—Whether or not irrigation should be done after the evacuation is a question which has been the subject of some discussion. The weight of opinion and experience condemns it, except in putrid effusions when the following may be used: boracic acid, saturated solution; bichloride of mercury, 1 to 5000 or 10,000; salicylic acid, 3 per cent.; hydrogen peroxide, 50 per cent. solution; pyoktanin blue, 1 to 1000 or 2000.

In making a choice of methods it is important to observe that patients even when very weak bear cutting well and, generally speaking, resection should be employed.

In conjunction with operative procedures the patient's strength should be maintained by nutritious and stimulating diet. Remedies, such as hepar sulphuris, silicia, calcarea, arsenicum, phosphorus, etc., as indicated, will prove useful adjuvants, and while they should always be administered they should only be regarded as such and never should be relied upon to the exclusion of operative interference.

After resolution every effort should be made to bring about expansion of the compressed lung and obliteration of the cavity; with this end in view, massage of the muscles of the affected side and breathing exercises should be practiced daily. The latter should occupy one or two minutes and should be taken three times a day. Ralston James' method has been employed with success. It is as follows: The patient is provided with two bottles connected with rubber tubing and holding a gallon each. One bottle is filled with water. There are also two tubes with mouth-pieces attached to the bottles. The patient blows through one of the mouth-pieces (the one attached to the bottle containing the water) and gradually transfers the contents by air-pressure to the empty bottle. Then the water is transferred back again. The effect of this procedure, as it may readily be seen, is to increase expiratory effort and systematically expand the lung.

4. Chronic Pleurisy.

While acknowledging that the clinical classification of pleurisy as acute and chronic has been questioned, and that the dividing line is often difficult to define clearly, there are certain types of the disease which are distinctly chronic.

The frequency with which pleuritic adhesions are found in autopsy is well known; in fact a large proportion of cases on post-mortem examination present some degree of pleuritic adhesions. Slight adhesions may form without giving any intimation of their presence.

Apart from empyema which has been considered from the acute and chronic standpoints and which generally follows a chronic course the same forms of pleurisy are recognized in its chronic manifestations as in its acute, namely, the plastic and the sero-fibrinous.

I. *Chronic Fibrinous Pleurisy.*—This variety presents two sub-varieties, viz.:

(1) Chronic or adhesive or primitive dry pleurisy.—This may follow the acute fibrinous form or it may originate insidiously as a chronic process without any preceding acute symptom. The plastic exudate, however slight, always tends to organize with the result of more or less adhesion between the layers of the pleura. Generally the adhesions are in limited areas and if tuberculous are usually situated at the apex and bilateral. In tuberculous cases small carious masses and tubercles are found in the thickened pleura. Sometimes the adhesions are extensive and general. Under these conditions the disease is unilateral. The different types of primitive dry pleurisy may best be described by quoting Sir Andrew Clark's classification, viz.:

"(1) Local or general uncomplicated adhesions. In the earlier periods of these formations the subjective symptoms are slight, there may be dragging, stitch-like pains on the affected side of the chest, especially on deep breathing. After existing for a period these symptoms may entirely disappear and the health remain unimpaired for an indefinite period.

"(2) A tuberculous variety which is an almost constant accompaniment of tuberculosis of the lungs. It is found generally at the apex where there are extensive adhesions and a more or less pronounced thickening of the pleura. In some instances this variety of tuberculosis pleurisy may be primary, that is, the infection may manifest itself in the pleura first and later may involve the lungs.

"(3) A non-tuberculous dry pleurisy characterized by excessive thickening of the pleura and the presence of fibrosis of the lung extending along the interlobular septa. This condition is sometimes called pleurogenic cirrhosis. The lung on section presents dense bands of fibrous tissue which extend from the thickened pleura deep into its substance. It occurs in middle and after life and is of infrequent occurrence. It is not accompanied by distinctive symptoms, but when long present there is failure of the general health and chronic bronchitis and sometimes bronchiectasis slowly develops. Under these conditions dyspnœa is often prominent and the urine albuminous. These pleurogenous types may also be complicated by dilatation of the right ventricle, failing compensation and dropsy."

(2) Chronic dry pleurisy following acute or chronic pleurisy with effusion.—In this form the fluid part of the effusion is absorbed leaving the fibrinous elements, which form connective-tissue adhesions between the two layers of the pleura. The latter are much thickened and cannot be separated. Generally this condition is more pronounced at the base of the lung, while the lung itself may become compressed and show the presence of more or less fibroid change. In a certain number of cases examination shows calcareous degeneration and small pockets containing remnants of the effusion. This condition as far as the resulting pathological changes are concerned more or less corresponds to the third type of Sir Andrew Clark's classification of primitive pleurisy.

The most pronounced form of secondary dry pleurisy is that which follows an old healed empyema when there is much retraction of the chest and hardening of the lung. Trauma, especially gunshot wounds, may be followed by the same results.

In most cases of secondary dry pleurisy there are few symptoms. There may be dragging pains in the affected side and some dyspnœa on exertion. The condition may continue indefinitely without injury to the general health.

PHYSICAL SIGNS.—The physical signs naturally vary according to the extent of the pleuritic process. *Inspection* in the milder forms, especially if the chronic process follows the acute, may show little or no visible change. In others there may be slight loss of motion. *Palpation* shows decrease or loss of vocal fremitus. *Percussion* may be negative or may give slight dulness. *Auscultation* gives no change or slight enfeeblement of the respiratory murmur. In the earliest periods a friction murmur may be heard.

In more severe cases, especially those which are the sequel of chronic sero-fibrinous pleurisy, the physical signs are more pronounced and are as follows: *Inspection* shows decided retraction. In rare instances there is unilateral sweating or flushing of the face and dilatation of the pupils due to complication of the first thoracic ganglion at the apex of the pleural cavity. *Palpation* gives absent or diminished vocal fremitus. *Percussion*, dulness, and if there is an excess of fibrous tissue the note may be of a tympanitic character. *Auscultation* shows the feebleness of the respiratory murmur and in some cases friction.

II. *Chronic Sero-fibrinous Pleurisy*.—This form also comprises two varieties, viz.:

(1) Primary chronic sero-fibrinous pleurisy.—This variety, sometimes called latent, develops as a primary chronic process. It comes on insidiously and apart from the physical signs may not be attended with symptoms. The fluid accumulates very slowly and the patient does not seek advice until he notices that his breath is short and his general health is below its former standard. Cases of this type are prone to be tubercular.

(2) Secondary chronic sero-fibrinous pleurisy.—This type develops after the subsidence of acute symptoms and repeated evacuations of the pleural cavity; the fluid persistently re-accumulates and remains for an indefinite period.

The symptoms are those of a mild degree of dyspnœa upon exercise, a somewhat compressible and quickened pulse and slight elevation of the temperature in the evening.

If the effusion becomes purulent symptoms of empyema will appear. The exudate in both varieties of chronic pleurisy with effusion does not differ from that of the acute forms except that it contains relatively less fibrin. As in the acute forms the effusion may cause displacement of the heart, liver and other organs. If absorbtion takes place there is more or less retraction of the affected side as in these cases the compressed lung never fully regains its normal tone.

The physical signs are the same as those of pleurisy with effusion.

The course of chronic pleurisy with effusion is from several months to several years. There is always danger of the development of pulmonary tuberculosis which terminates a large proportion of these cases. Another but less frequent complication is the change of the effusion into purulency.

Treatment.—The chief indications in the management of chronic pleurisy are the removal of fluid, if any be present, and the improvement of the patient's general condition. The former should be accomplished according to the rules discussed in the treatment of acute sero-fibrinous pleurisy. In all instances the patient should receive the most nutritious diet and should live out of doors as much as possible. In fact, the probability of a latent tuberculous infection should be always borne in mind. A mild climate and a moderate elevation are generally suited to most cases. Lung exercise should be practiced as described, including the method of Ralston James.

Remedies, although occupying a secondary position, are of much value. The chief are kali carb., sulphur, iodinum, arsenici iodidum, hepar sulphuris and silicia. Reference should also be made to other remedies mentioned in the discussion of the acute forms.

PNEUMOTHORAX.

Pneumothorax signifies air or gas in the pleural sac. It is very frequently associated with the presence of serum, pus or blood. These various conditions are expressed by the addition of the prefixes, hydro-, pyo-, and hæmo-, and their relation ætiologically to pneumothorax is designated by the position of the prefix in the compound word, thus, hydro-pneumothorax signifies serum in the pleural cavity subsequently complicated with pneumothorax; pneumo-hydrothorax signifies that the fluid follows the air; pyo-pneumothorax, pus in the pleural cavity followed by pneumothorax; pneumo-pyothorax, when the pus follows the air in the cavity. Thus, also, when blood is associated with the condition, it is expressed as hæmo-pneumothorax or pneumo hæmothorax. While it is well to know the full significance of these terms their clinical differentiation is not always possible or practical.

Ætiology.—Pneumothorax arises from the perforation of the pleura or from spontaneous generation of gases within the pleural sac in association with exudates. The latter is very rare. Perforation of the pleura may occur in the following conditions.

(1) Rupture of a tuberculous cavity into the pleural sac, or the breaking of a superficial focus of cheesy degeneration through the pleura.

(2) Diseases of the lung other than tuberculosis attended with necrotic processes, viz., gangrenous suppuration of the bronchial glands, septic or broncho-pneumonia, bronchiectasis, echinococcus cyst, cancer, rupture of a softened hæmorrhagic infarct with chronic disease of the heart.

(3) Empyema: In some instances perforation of the pulmonary pleura may result in the entrance of air into the pleural cavity; empyema necessitatis may likewise be a cause.

(4) Ulcerative processes from disease in other organs, such as cancer, abscess of the œsophagus, abscess of the chest walls.

(5) Perforation through the diaphragm in malignant diseases of the stomach, colon, etc. In these instances the causal lesions are all sub-diaphragmatic.

(6) Direct or indirect wounds, stabs, gunshot wounds, fracture of the ribs, etc.

(7) Violent muscular effort, such as lifting, violent coughing in pertussis. This can only occur when the lung is weakened by disease.

(8) Rupture of emphysematous air vesicles.

Spontaneous generation of gas within the pleural sac in associaciation with exudates, as a very rare phenomenon, may arise from the presence of the aërogenes capsulatus, the micro-organism to which the power of spontaneously evolving gases is attributed.

In regard to the frequency of these causes, the greater proportion of cases occur with pulmonary tuberculosis. The percentage being estimated from seventy to ninety. It may occur in both the acute and chronic forms, but is much more frequent in the latter. The perforation may be due to either the breaking down of an acute tuberculous focus, to the more gradual degenerative changes of a chronic process or to rupture of an emphysematous air-cell, especially when there are no pleural adhesions or thickening to protect the part. The accident usually occurs near the upper border of the upper or middle lobe, rarely at the apex and more rarely still at the lower border of the upper lobe.

The pathogenesis of pneumothorax is not alone dependent upon the perforation of the pleural membrane. There is another essential pathogenetic factor, namely, the condition of the tone of the lungs and of their co-aptability to the costal surface. Dr. O. Rosenbach of Breslau says: "It is evident that the living organism possesses an apparatus which reinforces the cōaptation of the pulmonary and costal pleura, as the functions of this apparatus are more perfect the better the state of the respiratory apparatus itself. It would seem almost as difficult to draw apart two glass plates as to separate the two healthy pleural surfaces by a foreign agent, as the wound appears to become at once hermetically closed by the vicarious activity (increased by expansion) of parts at some distance from the wound." That is to say, that in the instance of perforation of the pleura a cavity can only be formed between the pulmonary and costal layers when the tone of the lung, namely, its full capacity of expanding, is impaired. The force of this conservative influence is seen when it is considered how often in empyema, when after a free incision is made, the two surfaces of the pleura become completely adherent within a few days, or even within a few hours, provided the lung retains full ability of expanding and forced irrigation has not been employed. Furthermore, a wound in the lung is immediately followed by escape of air and collapse of the wounded air-cells. This acts as an obstruction to the further entrance of the air into the pleural sac. The accompanying hæmorrhage and extravasation and resulting inflammations also serve as obstructants.

Empyema necessitatis is rarely followed by pneumothorax, because the fistulous opening is so narrow and the rate of the discharge of pus is in proportion to the pressure within the pleural sac, that is, the expansion of the lung is usually in proportion to the evacuation of the pus, and thus keeps out the air. When, however, the evacuation of the pus is rapid and the opening is large, the lung can no longer act in this way, and in consequence the discharge of the pus is attended with the entrance of air into the pleural sac. Pnuemothorax is more frequent among males than females.

Morbid Anatomy.—The lesion may be situated on either side and as a rule the whole pleural cavity is distended. Circumscribed or partial pneumothorax may occur when there are old adhesions forming pouches in which the air is confined. This form is most frequent at the base of the lung, although it may occur at the apex. Double pneumothorax is extremely rare.

The mechanical effect of the distention is more or less obliteration of the intercostal spaces and displacement of viscera. When the pleural sac is punctured the imprisoned air rushes out with a hissing sound unless the pneumothorax is of the unusual form called open or the intra-pleural pressure is low. On opening, the pleural cavity may appear half empty and the viscera displaced, the degree of displacement depending upon the amount of distention. The pleural surfaces present the appearance of pleurisy and the lung is pressed against the spinal column, being reduced to one-fifth or less than its normal size. (See Fig. 56.) It is grayish or brownish in color when not covered with fibrinous deposits. On section it is found to be airless, hardened and full of blood. Many other changes in the lung are found which depend upon the initial lesion, as, for example, tuberculosis, septic broncho-pneumonia, etc.

In pneumothorax on the left side the heart and mediastinum are pushed towards the right. The heart, however, is not rotated and retains the normal relation of its parts. The diaphragm is depressed and the liver and spleen pressed downwards. According to experiments (Bartels, Fraentzel and Rosenbach) in artificial pneumothorax there may be considered bending and occlusion of the inferior vena cava.

Pneumothorax may be either open or closed. The former implies the free entrance and exit of air. Intra-pleural tension in this instance is the same as in the atmospheric air. The latter occurs when the opening is valvular and the egress of air is prevented or when the opening has become cicatrized. Intra-pleural pressure under these conditions may be that of the atmosphere or much higher. Sometimes the air may become absorbed and the tension become less or negative. A common type of pneumothorax is the valvular, that is, the opening is of such a formation that at certain times during respiration it allows the entrance of air, then closes. The point of rupture may be as small as a pin head or quite large. The former is more frequently observed in tuberculosis, the latter in extensive necrotic processes, such as gangrene, where it may appear as a tear or rent. When situated in the pulmonary pleura it may be found by placing the lung in water, inflating it and observing where bubbles of air appear. When in the costal pleura, the opening is usually easily observed. Sometimes there may be fistulous tracks between the pleural cavity and bronchi and in empyema necessitatis in the chest walls through the costal pleura.

The source of the distention of the pleural sac, except in rare instances of the presence of gas-generating bacteria, is the entrance from without of the atmospheric air. That which gains admission when the sac is closed loses its oxygen and carbonic dioxide increases, the proportion of the latter may be as high as 49 per cent. In open pneumothorax the air is practically the same as that of the external atmosphere. In putrid cases there is sulphuretted hydrogen.

Pleurisy and an exudate is almost always associated with pneumothorax. It may arise as the result of the primary lesion, in which case it precedes the entrance of air or it may follow the pneumothorax. In the latter instance the pleural inflammation is always the result of the irritant effects of the invading micro-organism, according to the type of which the exudate varies.

There is no reason to suppose that the contact of the atmospheric air with a serous membrane will cause it to inflame. It is the bacteria with which the air is laden which acts in this way. It was formerly thought that the exudate was generally sero-purulent, but since Senator's observation in 1888 it has been found that the serous exudate is more frequent than was supposed. A fibrinous exudate is rare. Blood may also be mixed with the exudate according to the nature of the same.

Stengel says that the air in pneumothorax may be absorbed and the pleura present no abnormality, but very frequently infection takes place and a purulent exudate forms.*

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* A Text-Book of Pathology, Alfred Stengel, Philadelphia, 1903.

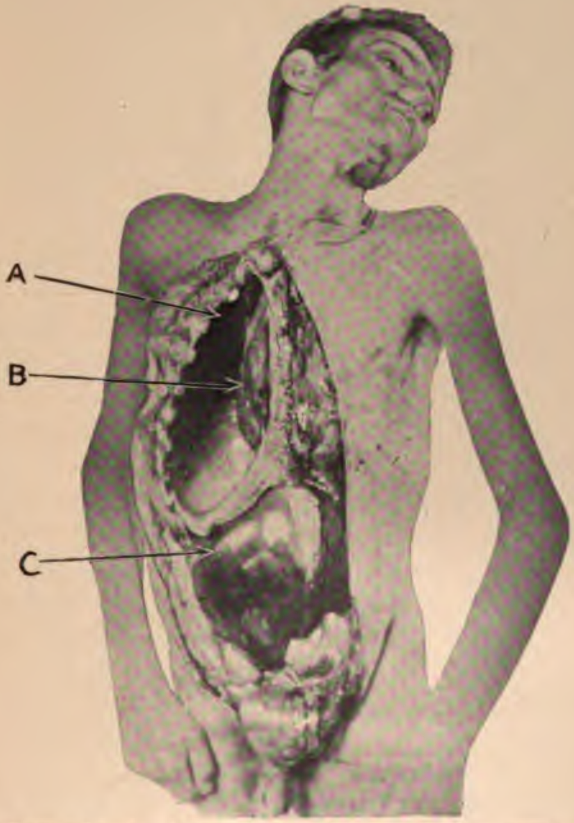

FIG. 55.—Pneumothorax. A, empty pleural cavity. B, lung compressed against spinal column. C, liver displaced downward. The case was one of pulmonary tuberculosis in which pneumothorax developed insidiously. Patient aged 25. Metropolitan Hospital.

In regard to the pathology of that very rare variety of pneumothorax which arises independently of the admission of air into the pleural cavity there does not appear to be a sufficient number of cases recorded upon which to base a positive description. It suffices to say that the gas generating micro-organisms, notably the aërogenes capsulatus, may give rise to the presence of gas in the pleural cavity. These organisms are not found in the healthy pleural sac, but in association with a decomposing exudate.

Symptoms.—The onset of pneumothorax is usually characterized by severe symptoms. There is generally sudden pain in the side, urgent dyspnœa, cyanosis, rapid, feeble pulse and great general distress. The face is expressive of anguish, the voice feeble or lost, the extremities cold and the temperature subnormal. The condition is one of collapse. Not infrequently the patient is aware when the rupture takes place. There is a sensation of something giving away accompanied by pain in the chest. Cough may be present, but as a rule is slight.

The upright position is usually sought. The patient may sit up in bed or on the edge bending forward and supporting himself with his arms, or may simply curve the back in a semi-prone position. The knees are always drawn up and sometimes the body turned on the affected side.

In exceptional cases the symptoms may come on with such suddenness and severity that fatal collapse soon follows. The intensity of the symptoms and the rapidity of their development depends upon the egress of the air. If little of the air which finds its way into the pleural sac during inspiration succeeds in escaping during expiration, which is the case when the opening is of valvular type, the cavity naturally fills with more or less rapidity, soon becomes distended and very serious symptoms quickly develop. If, on the other hand, there is some means of exit and the accumulation of the air is gradual, the symptoms are of a correspondingly milder type.

Frequently, even when severe, the early symptoms may gradually abate, the dyspnœa and pain become much more moderate and the temperature rise to normal, or above normal, owing to newly developing pleuritis. This improvement is often only relative, for the primary disease as a rule is rendered more unfavorable by the presence of the pneumothorax and the shock which accompanies it, so that the patient slowly sinks and in a few weeks succumbs to exhaustion.

There is a type of pneumothorax in which the condition is unattended with symptoms and where physical examination alone afford the only evidences of its presence. This variety is usually observed in debilitated persons whose systems have perhaps become more accustomed to a deficient supply of oxygen.

PHYSICAL SIGNS.—The signs of pneumothorax are striking. *Inspection* shows almost total immobility of the affected side, while in most cases there is considerable distention with obliteration of the intercostal spaces. The respiratory movements on the other side of the chest are exaggerated. The apex-beat of the heart may be seen in the left axillary region or to the right of the median line according to the seat of lesion and the amount of distention. *Mensuration* may be of service and the measurement of the two sides of the chest may be compared. *Palpation* shows diminution or loss of vocal fremitus, also the displacement of the apex-beat of the heart according to the side involved. The liver may be felt below the border of the ribs.

Percussion gives a variable note. It is usually loud and low pitched and is often described as resonant or tympanitic. The percussion-note may also be higher, resonant, as in emphysema, cracked-pot or even dull. These variations depend upon the degree of intra-pleural pressure and upon whether the muscles of the chest walls are relaxed or spasmodically contracted. The area of resonance may extend beyond the normal border of the lung. The loudness and depth of tone may be so marked as to cause the other or healthy side at first to appear dull. This percussion phenomenon is due to relaxation of the muscles rather than to the tension. As the accumulation of the air increases within the cavity the tension in consequence becomes greater, the sound loses its depth and becomes more shallow. In cases of excessive tension it also loses its loudness. Thus as the tension becomes excessive the pitch becomes higher until it approaches flatness.

Tympany occurs when the lung is retracted, the chest walls relaxed and there is a large accumulation of air in the anterior portion of the pleural cavity beneath the clavicle.

Cracked-pot resonance may be heard when there is a free communication for the entrance and exit of the air. Wintrich's sign may also be observed.

Percussion also discloses the displacement of organs. In right side pneumothorax the sudden and decided displacement of the lower border of the liver is an important sign. It can sometimes be felt at the level of the umbilicus in the median line. The displacement in pneumothorax is usually greater than in pleurisy with effusion, because of its sudden development.

When effusion occurs there is flatness, the line of which differs from that of ordinary effusion. The level of the latter adapts itself to the shape of the retracted lung, hence is curved. In pneumothorax the fluid no longer thus influenced by the lung assumes a natural level which constantly changes with the shifting position of the patient. Then the line of flatness changes.

Auscultation gives signs which are varied and at the same time of great importance. There is enfeeblement or complete loss of the normal respiratory and vocal sounds. When the breathing-sounds remain they frequently possess an amphoric or metallic quality. Sometimes the sound is low and musical, sometimes again, especially on coughing, the metallic tinkle of Laennec is heard. The point at which the metallic sounds are heard with greater intensity is below the clavicle, at the same time they may be heard over the lateral wall, in the supra-clavicular space and in some instances over the entire chest. The production of this phenomenon was supposed to be due to the dropping of fluid, but Skoda has shown that it may occur without fluid, for the sound of a râle or the bursting of a bubble of air may produce a note of a musical character when the latter is transmitted through a chamber of air.

The metallic character of the breathing-sounds is not always readily obtained on account of the lack of movement of the lung. To elicit it the patient should be directed to cough and thus set the air in the lung in vibration. The vocal resonance also has a metallic character. These metallic phenomena are especially marked in encapsulated or closed pneumothorax. In some cases the murmurs are loudly amphoric, both during inspiration and expiration, with a metallic quality. This is considered evidence of a free external opening.

Bronchial respiration may also be heard in portions of the lung which have undergone infiltration or consolidation. On the unaffected side the respiratory murmur is much exaggerated in marked contrast to that of the seat of pneumothorax.

Auscultory percussion gives the most characteristic phenomenon. If an assistant places a coin on the surface of the chest and taps it with another, and at the same time the examiner places the ear on the chest wall, a metallic sound is heard which is characteristic of pneumothorax and which was called by Trousseau, "*bruit d'airain.*" In rare instances the sign may be heard on ordinary percussion. In eliciting this phenomenon it is better to dispense with the stethoscope or phonendoscope.

Hippocratic succussion is a characteristic sign of pneumothorax when associated with effusion. Place the ear on the chest and shake the patient's body. A splashing sound is heard. Sometimes the splashing sound of succussion can be heard by the patient himself on moving and by others at a little distance.

In circumscribed pneumothorax the signs are the same except they are confined to limited areas, hence differ in degree. Circumscribed pneumothorax frequently is not recognized, especially in chronic pulmonary tuberculosis.

X-Ray Diagnosis, see Part VII.

Diagnosis.—Typical pneumothorax is readily recognized. The sudden development of dyspnœa with pain in the chest and symptoms of collapse, together with the physical signs of diminution or absence of the respiratory murmur, extension of percussion resonance more or less tympanitic in character over and beyond the border of the lung, displacement of the heart and diaphragm and above all the coin sign, can leave little room for doubt, especially so if there is a history of tuberculosis or other lung disease.

Too much importance should not be attached to tympanitic sounds, for the percussion-note it should be remembered is variable.

In those cases where the symptoms are not characteristic or when the condition comes on without giving rise to suspicion of its presence the diagnosis rests upon the physical examination alone.

In all cases of pulmonary tuberculosis frequent physical examinations are advisable for it is chiefly in this class of patients that pneumothorax is most likely to develop insidiously or without characteristic symptoms. In the circumscribed variety the difficulties of a diagnosis are much greater and a careful differentiation is necessary.

A condition most likely to be confused with pneumothorax is a very large pulmonary cavity. In both there may be tympanitic percussion, râles of metallic quality and distinctly amphoric respiration. In the rare instances when the lung is almost entirely excavated and the amphoric and metallic phenomena are very marked the liability to error is greater The main points of difference may be summarized as follows :

Pneumothorax.	Large Pulmonary Cavity.
Apex-beat of heart displaced, liver and diaphragm may also be displaced.	No displacement of the heart or other organs.
Vocal fremitus diminished.	Vocal fremitus not as a rule diminished ; generally increased.
Percussion-note loud and deep. Tympanitic-note not so common ; cracked-pot resonance and Wintrich's sign infrequent.	Percussion-note tympanitic, or cracked-pot ; Wintrich's sign generally present.
When effusion forms flatness over the same, the level of which changes with the position of the patient.	No flatness.
Respiratory sounds often absent ; amphoric respiration sometimes heard ; vocal resonance absent.	Respiratory sounds are cavernous and amphoric ; there is also pectoriloquy, gurgling and crackling.
Coin sound present.	Coin sound exceedingly rare.
Hippocratic succussion-splash.	No hippocratic succussion.

Subphrenic abscess containing air may simulate pneumothorax. The points of difference are as follows : It is preceded by the history of abdominal disease, which is rarely the case in pneumothorax, the chief causes being ulcer of the stomach or duodenum followed by localized peritonitis, perforation and the formation of abscess immediately beneath the diaphragm ; again, there is only slight displacement of organs ; the physical signs are confined to the base of the lung, while the apex and upper portion remain normal or nearly so ; there is often bulging of the hypochondrium and depression of the liver ; cough and expectoration is absent and lastly there is Pfuhl's sign. This is the effect of an exploratory incision or puncture. In subphrenic abscess the liquid which issues comes forth with considerable force during inspiration, whereas in pneumothorax the contrary occurs.

Diaphragmatic hernia is another rare condition which may prove a possible source of error in the diagnosis of pneumothorax. A differentiation may be made by noting the following features: Diaphragmatic hernia, while it may be congenital, is usually the result of some severe crushing injury; again the hernia may recede and all symptoms suddenly disappear and re-occur with sudden return of symptoms. The third point of difference is the presence of a rumbling sound peculiar to the bowels. In other respects the two conditions present similar symptoms and signs.

Emphysema scarcely seems a possible source of error and could only prove such through oversight or carelessness. Emphysema as a rule is bilateral, its onset is slow and insidious, covering several years; the formation of the chest is characteristic; it is not attended with pain or shock, nor with the physical signs of metallic sounds, succussion-splash or the coin sound.

A partial pneumothorax at the extreme base of the lung posteriorly is difficult to diagnose from a small pleurisy with effusion. It may be attended with a flat percussion-note and diminution or absence of the respiratory murmur. In such cases the only means of diagnosis is exploratory puncture.

Pleurisy with effusion may sometimes exhibit hyper-resonance above the fluid and may in consequence suggest pneumothorax, but it always fails to exhibit the physical phenomena which characterize the latter.

Gaseous distention of the stomach is also mentioned as a possible source of error in diagnosis, but a careful examination of the history of the case and the results of treatment directed to the alimentary tract will remove all doubt.

Prognosis.—The prognosis depends for the most part upon the cause. When secondary to pulmonary tuberculosis the greater proportion of patients die. This is largely due to the fact that tuberculous pneumothorax, as a rule, does not occur until the tuberculous process is well advanced and the resistive powers of the patient are much enfeebled. Many are unable to recover fully from the effects of the shock which so frequently attends the entrance of air into the pleural cavity, but simply rally for a short time and succumb at the end of two or three weeks. Immediate death is uncommon. When the general condition is good life may be prolonged several months. In a small proportion of cases recovery takes place.

The development of pleurisy and febrile reaction may be regarded on the whole as rather favorable, and if, after about ten or fourteen days the fever subsides and only a moderate amount of serous or sero-purulent effusion is present, the patient may be regarded as doing fairly well. He may remain in this condition for several months or even longer and eventually die from the tuberculous infection.

When the effusion is purulent the prognosis is necessarily more unfavorable. Putridity of the exudate is also an unfavorable indication. Double pneumothorax while excessively rare is generally fatal within a few hours.

Glancing at the ætiology it will be apparent, with a few exceptions, that pneumothorax from other causes holds out little hope of recovery on account of the nature of the primary lesion. A notable exception is traumatism in healthy persons, especially if the patient is young; under these conditions recovery is frequent.

Accidental rupture of the pleural sac in chronic emphysema is decidedly unfavorable. Pneumothorax with abscess of the lung in some instances is followed by recovery.

Cases which go to resolution require from one to two months for the absorbtion of the air and exudates.

Circumscribed or partial pneumothorax does not effect the course of the primary disease unless the exudate is purulent.

Treatment.—In cases attended with severe symptoms, pain, dyspnœa and collapse, morphine, one-quarter of a grain should be immediately administered. It not only relieves the pain but the shock as well and should generally be preferred. Heat should be applied to the extremities and seat of pain in the form of hot water bags. Hot fomentations may also be used with advantage. If collapse seems imminent and the heart threatens to fail, strychnine hypodermically should follow the morphine. Other stimulants, such as whiskey, brandy or ether, may also be used. If the case is one in which the opening is valvular and the air enters and accumulates, causing distention with intense dyspnœa and distress, the tension should be relieved by evacuation. For this purpose the ordinary trocar and cannula should be employed under the usual antiseptic precautions. The latter should be retained *in situ* until it is certain that the air will not accumulate again. The cannula should be held in position by plasters and covered with antiseptic dressings.

The exudate in pneumothorax, if serous and not excessive, does not call for interference. Even when it is considerable, aspiration should be delayed. The early performance of aspiration should, in fact, always be deprecated, as at this period of the disease the perforation is likely to be patent or at the best imperfectly closed. A good general rule is to wait at least six weeks, unless the patient is suffering much distress or life is endangered, when it should be resorted to without delay and without relation to the onset of the pneumothorax.

When there is an excess of air over the amount of exudate, Weil suggested that the former be withdrawn to the amount of 400 to 1000 c.c. This is certainly reasonable, for it is recognized that air in the pleural sac is difficult of absorbtion.

In purulent exudates the methods of procedure vary according to the primary condition and the demands of each case.

In tuberculous cases incision is frequently attended with unfavorable results. Its application should be limited to patients in fairly good condition in whom there are reasonable grounds of hope for a considerable prolongation of life and a possible arrest of the disease. In advanced cases with emaciation and cavities, aspiration should be preferred and repeated as often as necessary. In cases of this type incision is contraindicated.

The treatment of traumatic pneumothorax does not call for any special line of consideration, apart from that discussed. In cases of this nature there is one point to which it is important to call attention, that is, absolute rest in bed. After the subsidence of the early symptoms there may be no pleurisy, and in consequence the patient may feel quite well. Rest in bed, however, should be strictly enforced. In circumscribed or partial pneumothorax the treatment should be on the same general plan as in the other forms of the disease.

The following case of pneumothorax, due to puncture of the pleura from fracture of the rib, is typically illustrative:

A boy of seven was run over by a wagon on the morning of June 14, 1904, and removed to the Flower Hospital. Examination by the ambulance surgeon shortly after the accident revealed a slight abrasion over the right sixth rib. The patient was moaning, greatly frightened and complained of pain low down in the abdomen. His face was flushed, respiration good, pupils normal. He was placed in the ambulance and immediately sank into a condition of serious collapse. Respirations became labored and faint, pulse almost imperceptible, skin pallid, cold and covered with perspiration, eyes rolled upward and pupils unequally dilated. Strychnine, gr. $\frac{1}{60}$, was administered hypodermically, and on arrival at the hospital the patient was placed in bed, where he revived. He now complained of pain through the abdomen and chest, was restless and unable to urinate. The respirations were 44, pulse 124, rectal temperature 98.2° F. In the evening, respirations were 60, pulse 144, rectal temperature 98.6° F. There was frequent urination, difficult deglutition, and the patient lay with his knees flexed. He suffered from sudden paroxysms of severe pain in the region of the anterior attachment of the diaphragm, namely, the ensiform cartilage and the fifth and seventh ribs. During these paroxysms he would cry out as if in great suffering, the abdomen would become rigid, the surface bathed in profuse perspiration, the lips cyanosed and the face flushed. These attacks would last for several minutes.

Physical examination revealed the following: Inspection: immobility of the right side of the chest with partial obliteration of the intercostal spaces; exaggeration of respiratory movement on the left side and displacement of the apex-beat of the heart to the left axillary line. On further examination fracture of the sixth rib on the right side was discovered. Palpation: loss of vocal fremitus on the right side. Percussion: hyper-resonance, with some tympany on the right side. Percussion also confirmed the displacement of the heart to the left, and in addition showed the liver to be pushed somewhat downward. Auscultation: marked exaggeration of the breathing-sounds of the left side, great enfeeblement of the same sounds on the right, and sharp metallic sounds on coughing over the right lung. Auscultatory-percussion: the coin test with the metallic sounds typically.

It was evident that the sharp extremity of the fractured rib had punctured the lung. The wound to the lung was doubtless inflicted at the time of the run-over accident, but from the history of the case actual perforation was not complete and the air did not enter the pleural cavity until the patient was placed in the ambulance, when the symptoms of collapse appeared. From the 15th to the 18th of the month, inclusive, respiration varied from 60 to 72, remaining most of the time at 60; the pulse from 130 to 146; the temperature 99.8 to 100° F.

The paroxysms of pain gradually grew shorter and less frequent. On the 19th, the respirations fell to 48, the pulse and temperature remained the same. On the 23d, the respirations showed a tendency to fall, the pulse remaining stationary. On the 24th, the pulse was 108, respirations 44, temperature 99.2° F.

From this time there was a decided tendency to return to the normal condition. The physical signs, however, remained the same, only less pronounced. The paroxysms of pain had now entirely disappeared.

On June 30th the respirations were 24, pulse 96, temperature normal. The patient felt well and his parents insisted upon taking him home.

The treatment, after the recovery from the shock, consisted simply of the application of an adhesive strap over the fractured rib, absolute rest in the recumbent posture and low diet. It was obvious that no interference was called for.

The paroxysms of pain, especially when taking into consideration the crushing nature of the injury, in the opinion of the writer, was due to hernia of the diaphragm.

SUBPHRENIC PYO-PNEUMOTHORAX.

This term was applied by Leyden to that uncommon form of abscess situated in the abdominal cavity beneath the diaphragm, which becomes distended with gas from the intestines or stomach and presses upward upon the pleural sac and lungs. It is not a true pneumothorax as the gas does not find entrance into the pleural cavity, but the symptoms and effects upon the lung are in many respects similar.

These abscesses may arise in connection with any process which may cause perforation of the abdominal viscera, namely, peritonitis, gastric or duodenal disease, carcinoma, hepatic or splenic abscesses, decomposing echinococci, rupture of the gall bladder, and as the sequel of infection from pleurisy. The abscess having formed, gas from the intestines or stomach finds entrance and distends its cavity. If there is no chance for the sac to expand in the downward position on account of adhesions, it naturally pushes upwards, encroaches upon the pleural cavity and presses against the lung. The result of this condition is a train of physical signs which may simulate those of pneumothorax, viz.: loss of fremitus, metallic phenomena and loss of respiratory murmur. Other symptoms are milder pain on pressure, absence of bulging of the intercostal spaces and tension of the muscles with slight displacement of the organs. There is also the history of abdominal disease, such as mentioned in the ætiology, which affords material aid in the diagnosis. There is no cough and the normal vesicular breathing may be audible over a large part of the lung, while that part which is compressed by the abscess is usually clearly defined and on auscultation gives amphoric breathing.

HYDROTHORAX.

SYNONYM.—*Dropsy of the Pleura.*

Hydrothorax signifies the presence of fluid in the pleural cavity of non-inflammatory origin.

Ætiology.—It is always a secondary affection and generally arises in connection with cardiac, renal or hæmic diseases where it constitutes part of the general dropsy. It may, however, occur independently of the latter. Osler mentions a case of post-scarlatinal nephritis in which hydrothorax was the sole manifestation of dropsy except for slight œdema of the feet and ankles. This fact should not be overlooked and in all cases of nephritis the presence of dyspnœa should suggest the possibility of hydrothorax and examination of the chest should be made accordingly. When of cardiac or renal origin it appears late in the course of the primary disease and may prove the terminating complication. Cases of hæmic origin occur in conditions of profound impoverishment of the blood and are found associated with pernicious anæmia, leukemia, malaria, syphilis, scurvy, chronic diarrhœa, chronic dysentery and cancer. In exceptional instances the condition may be due to local causes, such as the pressure of tumors or aneurysm so situated as to impede the return flow of blood from the pleura or to press upon the superior vena cava or thoracic duct.

Morbid Anatomy.—Hydrothorax is usually bilateral, but in cardiac disease it may occasionally be unilateral when there is much enlargement of the heart. In such cases it is generally on the right side. This is probably due to pressure upon the vena azygos and veins of the root of the right lung. It may also be unilateral when due to the pressure of tumors or aneurysm.

The transuded fluid is clear amber-colored with alkaline reaction and low specific gravity, 1009 to 1012. It consists of water, albumin and salts, but contains no fibrin. The amount varies from a few ounces to several pints, but such excessive quantities are rare. The pleural surfaces are smooth, free from fibrinous exudate, pale with loss of lustre and opacity and sometimes œdematous. In rare instances the fluid may be encapsulated owing to the presence of old pleuritic adhesions. There is compression of the lung and displacement of adjacent viscera as in pleurisy with effusion. A small amount of fluid often transudes into the pleural cavity immediately before death.

Symptoms.—As in all secondary affections the prevailing symptoms of the primary disease appear with more or less prominence. The symptoms which belong to hydrothorax, it is needless to say, vary according to the amount of exudate present in the pleural sac and the degree of compression which results. The most prominent is dyspnœa. This may vary from mild breathlessness on exertion to conditions of agonizing orthopnœa. A feature of the breathlessness is its increasing intensity. There is also irritative cough and asthmatic attacks accompanied by cyanosis, cold perspiration and every evidence of great distress. Fever and local pains are absent.

PHYSICAL SIGNS.—The physical signs are the same as those of pleurisy with effusion with the exception that they differ in degree and in the fact that pleural effusions are unilateral and the level of the fluid is curved, while hydrothorax is usually bilateral and the level of the fluid follows more closely natural gravity. The displacement of viscera is also less, not only because the amount of fluid is generally less, but because the abdominal cavity is usually also distended with fluid and in consequence exerts an upward pressure. In bilateral hydrothorax the pressure on the two sides is more nearly equal.

X-RAY DIAGNOSIS, see Part VII.

Diagnosis.—The signs of fluid in the pleural cavity, especially if on both sides, in association with cardiac, renal or hæmic diseases and dropsy in other parts, leave little doubt of its non-inflammatory origin.

Unilateral hydrothorax at first glance may simulate pleurisy with effusion, but a closer examination will scarcely permit of confusion, as it is generally observed in connection with advanced diseases of the heart with much enlargement and failing compensation. There is also the difference in the line of level of the fluid in the two conditions as stated in the physical signs. For further points of difference, see Diagnosis of Pleurisy with Effusion.

Prognosis.—The nature of the primary diseases with which hydrothorax is associated naturally renders the outlook unfavorable. Again, inasmuch as it does not usually develop until the causal lesion is far advanced, its presence must be regarded as of unfavorable significance. The fluid may be removed by the ordinary process of aspiration, but it soon re-accumulates. A moderate amount of fluid may be tolerated by the patient without serious inconvenience, but excessive accumulation causes death. Filling of the chest may prove fatal in two or three days.

Treatment.—The treatment of hydrothorax necessarily must be directed to the cause, but as this is incurable it is at the best only temporary in its effect. Cases depending upon cardiac, renal or hæmic disease must be managed on such lines as these affections demand. In cardiac and renal diseases the action of the heart and that of the kidneys should be stimulated in order to free the tissue of the water. The diet should be dry as possible, the bowels should be kept free. Purgatives may be of service, but should be used with caution, if at all, lest they weaken the patient. Aspiration should not be performed except when the dyspnœa is distressing, when it should at once be resorted to in order to afford immediate relief. The remedies which will prove of most service are the infusion of digitalis, apocynum, apis, infusion of triticum repens, arsenicum and the infusion of scoparius. For other remedies the reader is referred to the article on dropsy and the discussion of the treatment of valvular diseases of the heart.

In hydrothorax of hæmic origin the treatment is that of the blood disease itself.

HÆMOTHORAX.

Hæmothorax signifies the presence of blood in the pleural cavity without inflammation.

Ætiology.—The condition arises secondarily as the result of pathological processes or the direct effects of trauma. The former includes rupture of an aneurysm, cancer of adjacent structures, necrosis of the rib causing erosion of an intercostal artery, rupture of a pulmonary infarction, the hæmorrhagic diathesis, scorbutus and cirrhosis of the liver. Hæmothorax of pathological origin is very unusual, hence its presence with any of the aforementioned morbid conditions is rare. Trauma is a more frequent cause. Any wound which lacerates the pleura and intercostal arteries is necessarily attended with more or less hæmorrhage into the pleural cavity.

Morbid Anatomy.—Apart from the pathological changes incident to primary disease and the extent and character of the wound in cases of traumatism, the question in the morbid anatomy of hæmothorax is the effect of the presence of blood in the pleural cavity and the changes which take place in the blood itself when in that locality. As regards the first, more or less pressure upon the lung and displacement of viscera results as in effusion from pleurisy. The changes which occur in the blood have been the subject of experimentation, the conclusion being that the effused blood resists coagulation to a considerable degree and that if the pleura is healthy and infection by pyogenic organism does not take place, absorbtion is rapid. When, on the other hand, the pleura is diseased or infection occurs, pleurisy, suppuration and changes in the blood occur.

Symptoms.—In certain cases, such as aneurysm of the aorta, death is instantaneous. Under other conditions the symptoms vary according to the amount of the hæmorrhage. If very slight there may be little disturbance, but if at all copious there is pallor, rapid anæmia, feeble pulse, coldness of the extremities and marked dyspnœa. Sometimes there is sudden intense pain and syncope. Dyspnœa is a prominent symptom and is always excessive when the hæmorrhage is large. The rapidity with which the hæmorrhage occurs also influences the symptoms ; a sudden pouring out being attended with abruptness of onset and sudden intensity of symptoms, whereas if the bleeding is gradual the symptoms are much slower in developing. The subsequent course of hæmothorax varies according to the nature of the primary lesion and presence of infection. If the patient survives, pleurisy, pneumothorax with suppuration and putrid decomposition are frequent complications. The physical signs are those of pleurisy with effusion.

Diagnosis.—The history of the case, the symptoms of internal hæmorrhage and the signs of fluid in the pleural cavity which have suddenly made their appearance point conclusively to the condition. There is usually some difficulty in locating the source of the bleeding. The presence of hæmoptysis suggests the lung as the seat, but its absence on the other hand does not prove the contrary. After the lung, the intercostal and mammary arteries are the most frequent sources of bleeding.

Prognosis.—The immediate danger to life in hæmothorax when the bleeding is copious is very great, and if a vessel of any size is involved the condition rapidly proves fatal. If the patient survives the first shock and the bleeding is arrested there is always danger from further hæmorrhage and the subsequent complications which are likely to follow. Again the primary lesion from its very nature tends to infection.

In those instances where there is no infection the outlook is more hopeful as the blood is absorbed rapidly. This favorable condition is generally observed in comparatively mild cases of trauma.

Treatment.—To arrest the hæmorrhage, which it is needless to say is the first indication, apply ice-bags to the chest anteriorly, between the scapulæ posteriorly and to the scrotum, and administer morphine hyperdermically.

No attempt to remove the blood should ever be made unless there is an excessive degree of compression and urgent symptoms arising therefrom, when an amount of the effusion sufficient to relieve should be withdrawn by aspiration ; or unless there is suppuration. Further indications of treatment must be met by the individual demands of each case which in trauma are mainly surgical.

CHYLOTHORAX.

Chylothorax signifies chyle in the pleural cavity. The term chylous pleurisy is not synonymous and should not be confused with it. Chylous or more correctly chyloform pleurisy is simple pleurisy in which the effusion is of a milky appearance due to the fatty degeneration of cells. It is therefore pathologically far removed from chylothorax.

True chylothorax is a leakage of chyle into the pleural cavity. It is very rare and but few cases are on record. It arises from rupture or obstruction of the thoracic duct in any part of its length or rupture of a large lymph vessel within the thorax due to trauma, new growths, occlusion from thickening or tuberculosis. It may be unilateral or bilateral. In some instances when the former, there is a limpid effusion on the other side. In a case reported by Dr. Turney the effusion was chylous on the right side and chyloform on the left. In a large proportion of cases chylous ascites is an associated condition. The symptoms are those of the primary lesion with the addition of more or less dyspnœa and pain in the side. The pulse shows some acceleration and the temperature is negative. The physical signs are those of pleurisy with effusion.

The diagnosis depends upon the results of exploratory aspiration and cannot be made otherwise with any degree of certainty. Examination of the fluid should be made in order to distinguish the condition from pleurisy with chyloform effusion, which might be mistaken for true chylothorax. This, however, could only occur in cases of pleurisy of the chronic type, for the history of the case would otherwise prevent any possibility of error.

The prognosis is very unfavorable. Anastomosis with the right duct may take place in cases of slow development. Death usually occurs inside of a year.

Treatment consists in relieving symptoms and in supporting the patient's strength by nutritious diet and proper hygiene. If the chyle in the pleural cavity ever becomes so excessive as to give rise to urgent symptoms aspiration should be performed.

SYPHILIS OF THE PLEURA.

Syphilis of the pleura is a condition in reference to which it is difficult to speak with any degree of certainty. Pleurisy in the neighborhood of gummata may reasonably be regarded as specific. Pleuritic thickening in syphilitics, as in the instance of pulmonary consolidation under similar conditions, may also be of specific origin.

NEOPLASMS OF THE PLEURA.

Several varieties of tumors are found in the pleural cavity, but none are in any way peculiar to it. Fibromata may be observed in the serous or sub-serous layers of the costal or visceral pleura where they occur as nodular masses ; chondromata, lipomata and even osteomata are also found. But all of these morbid growths are so rare that they do not call for more than mention. The most frequent forms of neoplasms of the pleural membrane are carcinomata and sarcomata, the former constitutes so large a proportion of cases that it will be considered almost exclusively.

Carcinomata of the pleura may be either primary or secondary. The existence of the primary variety was formerly denied, but Wagner in 1876 described what he called primary endothelial cancer of the pleura. His observations were subsequently verified by Fränkel, Neilson and others. While very unusual, a number of cases are on record. The disease presents certain special features, the chief characteristic being thickening and increased density of the pleural membrane. The thickness may sometimes measure from one to two centimetres or even more. In appearance the pleura resembles fibroid thickening rather than a neoplastic growth and may be mistaken for the same.

The surface is either smooth or trabeculated. Adhesions are also usually present. On section a few drops of milky fluid may be pressed from the cut surface. Under the microscope the changed tissues are found to consist of a fibrous stroma with large lymph spaces containing polymorphous cells. Fränkel regards these changes as better described under the name of "lymphangitis prolifera," at the same time admitting their malignancy.

In cases of considerable duration the chest appears contracted, especially laterally and posteriorly. This is due to the contraction of the thickened pleural tissues. A striking feature is the rapidity with which this change takes place. The lung may undergo shrinkage, becoming atelectatic, or it may share in the malignant process, or it may on the other hand show little or no change.

Secondary cancer is the usual form of malignant disease of the pleura. It may arise by metastasis from extension of cancer of the breast, axilla, œsophagus, mediastinum, stomach or other adjacent structures.

The pathological changes are variable. The pleural cavity may be simply invaded by a cancerous growth from a neighboring organ, or the pleural membrane may be studded with disseminated nodules, sometimes prominent, sometimes flattened, varying in size from a pea to from four or five inches in diameter. When the growth is large there is necessarily displacement of the heart and compression of the lung.

Primary sarcomata of the pleura is also very rare; when observed it is more frequent in children and generally of the spindle-cell variety.

Secondary sarcomata may also arise by metastasis or from extension from adjacent organs.

Sero-fibrinous pleurisy usually accompanies cancerous formations; the effusion may be simply serous, but is generally hæmorrhagic. In a certain proportion of cases the effusion has been found to be chylous.

Symptoms.—The symptoms of cancer of the pleura develop very gradually and in the early periods of the disease are not well defined. The patient usually first complains of dull pain in the side, later this becomes more pronounced and generally pleurisy with fever develops, as a rule, followed by effusion which is often large.

Primary cancer of the pleura may simply present the symptoms of chronic pleurisy with or without effusion. Pain, however, is apt to be more prominent. If the growth attains large size there is proportional pressure upon the lung and dyspnœa. There may also be evidences of a tumor in the pleural cavity. In addition to these symptoms there is the progressive emaciation, greenish-yellow skin and loss of strength which characterizes malignant diseases.

In some cases the early symptoms appear suddenly simulating an attack of acute pleurisy with all the phenomenon of that condition. This mode of onset is exceptional.

PHYSICAL SIGNS.—The physical signs are variable. Enlargement of the cervical and axillary glands is common. In secondary cancer the primary lesion may be in evidence. Large pleuritic effusions mask the morbid growths in some cases, especially those of the endothelial variety. Retraction of the lateral and posterior portions of the chest wall is pronounced. This feature is noticeable after aspiration as well as in cases where the amount of effusion is large. In other instances there may be evidences of compression of the lung with a circumscribed area of bulging of the thoracic walls. Over the tumor itself the percussion-note is flat, fremitus is absent or diminished; there is also on palpation a feeling of resistance and the respiratory murmur is diminished or absent.

X-RAY DIAGNOSIS, see Part VII.

Diagnosis.—In cases where there are evidences of malignant growths elsewhere, the detection of an area of circumscribed flatness in the thoracic region with diminished vocal resonance and respiratory murmur should excite the suspicion of implication of the pleura. Again the presence of pleurisy with effusion under the same conditions should also raise the same question.

The recognition of primary cancer of the pleura is very difficult. It may simulate chronic tuberculous pleurisy with effusion or pulmonary consolidation. In many instances its presence is not even suspected until exploratory aspiration is performed, when the decidedly hæmorrhagic character of the exudate directs attention to the possibility of the existence of either malignant or tuberculous disease. This point may be definitely settled by examining the effusion for small masses of cancerous tissue which may be found if the growth is breaking down. There are two other points of difference, first, the presence of fat, either free or contained in degenerated endothelial cells, the latter being sometimes so abundant as to give the cells a mulberry appearance. Second, the fact that the blood in the malignant effusion of cancerous pleuritic disease is darker in color than in that of tuberculous pleurisy. It should not be forgotten, however, that in both the effusion may be serous. Again in cancer pain is always more prominent than in chronic pleurisy. The cachexia should also excite suspicion of the true condition.

From pulmonary consolidation cancer is to be distinguished by the presence of pain, the circumscribed area of flatness rather than dulness with the resistive feel, the cancerous cachexia, and on aspiration the presence of cancerous tissue in the exudate and effusion.

Prognosis and Treatment.—The duration of life is from six to twenty months. Treatment is purely palliative. Aspiration may be performed if the effusion is sufficientiy large to cause urgent symptoms. It tends, however, to rapidly re-accumulate.

HYDATIDS OF THE PLEURA.

Hydatid cysts of the pleura may be either primary or secondary. In both instances they are very rare. The rarity of primary cysts may be judged from the statement of Whitney* that of nineteen hundred cases collected by Thomas only one per cent. involved the pleura alone, and out of nine hundred and eighty-three cases Neisser found that only seventeen were primary.

Secondary hydatids of the pleura, which constitutes the usual form of the lesion when observed, arise most frequently from primary growths situated in the liver, and are therefore seated at the base of the right lung. Frequently a primary cyst of the lung may discharge small daughter cysts into the pleural cavity. According to Trousseau this is the usual form of pleural hydatid.

Morbid Anatomy.—Post-mortem examination shows that the cysts are generally single and consists of a tumor which occupies more or less of the pleural cavity. In structure the cysts are the same as when situated elsewhere in the body, but are peculiar in the fact that the fibrous tissue surrounding the sac is very thin. The size varies with the age of the cyst. If it has existed long it may contain three or four pints, and cause almost complete collapse of the lung; as much as nine pints have been removed.

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* Twentieth Century Practice of Medicine, vol. vii.

As a rule the cysts merely compass the lung, but they may penetrate and discharge through the bronchi.

Symptoms.—In most instances there are few symptoms until the cyst becomes sufficiently large to interfere with respiration, in others again pain appears with the onset of the disease. A feature of the pain is its persistence throughout the entire course of the affection. Later, dyspnœa develops and increases greatly with the growth of the cyst. Cough may be present, but it is not so prominent as in hydatids of the lung. It is dry and generally without hæmoptysis, the latter being rare. Emaciation and anæmia are generally steadily progressive. Fever is usually absent, unless there are complications.

PHYSICAL SIGNS.—The physical signs are dependent upon the size of the tumor and the integrity of its walls. If of some size and the walls are intact, the following signs appear: some bulging of the affected side which is of a globular nature and which varies with the size of the cyst, occasional fluctuation of the intercostal spaces, loss of vocal fremitus, flatness on percussion and diminution of the respiratory murmur. There may also be displacement of the diaphragm and mediastinum. Hydatid fremitus is absent. These signs are in no way distinctive and may be observed in encapsulated pleurisy with effusion and solid tumor.

Diagnosis.—The absence of distinctive symptoms renders recognition of pleural hydatids a matter of great difficulty. A positive diagnosis depends upon the recognition of the echinococcus hooklets in the fluid withdrawn from the pleural cavity. The fluid of the hydatid or echinococcus cyst has a specific gravity of 1009 to 1015, is clear, neutral or alkaline in reaction, contains no albumin, or only faint traces, and a considerable amount of sodium chloride. Sugar is sometimes found. The clearness of the fluid and absence of albumin points to hydatids, but is not conclusive. Careful search must be made for the echinococcus hooklets.

The similarity of the physical signs to those of incapsulated pleurisy and solid tumor is liable to prove a source of error unless there are definite features in the history which enable a conclusion to be reached. Points of difference in regard to pleurisy are that hydatids of the pleura are often situated at the base of the lung, and the bulging of the chest walls has the globular character as mentioned, but neither of these features are conclusive and the diagnosis will depend always upon the recognition of the echinococcus hooklets.

It is often impossible to differentiate hydatids of the pleura from that of the lung, even when two or three ribs have been removed, for a pulmonary hydatid may reach the visceral pleura and become fused subsequently with the parietal layer, so that when incision is made no pleural cavity is found, but the knife passes directly from the parietal pleura into the cyst itself.

Prognosis.—This is very grave. If the case is left to nature the patient dies in from one to a little over four years.

One of the principal dangers is rupture of the cyst into a bronchus with asphyxiation of the patient from inundation of the lungs by the hydatid fluid. If the patient escapes this accident pneumopyothorax usually results and the physical signs vary accordingly.

Operation renders the outlook much more hopeful.

Treatment.—The management of hydatids of the pleura is surgical. Aspiration is contraindicated and should be employed solely for diagnosis and to afford temporary relief. The only course which promises recovery is the radical removal of the growth by resective thoracotomy. Whatever operation is decided upon it is important that it should be performed early.

PART IV.

DISEASES OF THE MEDIASTINUM.

DISEASES OF THE MEDIASTINUM.

THE principal affections of the mediastinum, apart from those of the great organs which it contains, are inflammation of its tissues, enlargement of the glands, abscess, tumors and hæmorrhage.

INFLAMMATION OF THE MEDIASTINAL TISSUES AND BRONCHIAL GLANDS.

Inflammation of the mediastinum may involve the serous surface of the duplicature of the pleura which separates the pleura from the mediastinal cavity, the connective tissue and glands of the mediastinal space. The former is, strictly speaking, a variety of pleurisy, pleuro-mediastinitis, and as a rule cannot be distinguished during life. The inflammation may likewise result from extension in pericarditis (mediastino-pericarditis). There may be signs pointing to the condition, but it is always difficult and often impossible to recognize it.

The inflammation may likewise involve the bronchial glands and connective tissue and may arise either primarily or secondarily. Primary inflammation may result from trauma, accidental or operative, or from exposure. It is not common. Secondary forms are more frequent and occur in association with bronchitis, pneumonia, whooping-cough, pleurisy, tuberculosis, the infectious fevers and syphilis.

Inflammation of the bronchial glands which has been described by some writers as a separate entity is practically a secondary affection and one generally impossible to recognize except at autopsy.

The presence of persistent cough for which no cause can be assigned is said to point to its presence. In tuberculosis the bronchial glands often share in the infection undergoing inflammation enlargement, suppuration and caseous degeneration.

A mild degree of inflammation of the bronchial glands and simple lymphadenitis is common in all forms of bronchial and pulmonary inflammations ; when associated with measles or whooping-cough the inflammation is frequently severe. It has been claimed that enlargement of the glands in the posterior mediastinum is responsible for the cough in whooping-cough.

The bronchial glands may also become enlarged in leucocythemia and Hodgkin's disease as part of the general process.

It is stated that enlarged bronchial glands can be recognized by physical signs. This could only be possible when there is dulness or percussion appreciable in the upper inter-scapular region posteriorly, or in the region of the manubrium.

ABSCESS OF THE MEDIASTINUM.

Abscess of the mediastinum may be either acute or chronic. In both instances it is rare.

Acute abscess may arise from any of the causes which may induce inflammation of the mediastinal structures, especially trauma and the acute infections, such as erysipelas and variola, the latter being the most common. Again it may originate from suppuration in adjacent organs.

Sometimes the suppurative process is diffuse. The symptoms are generally described as consisting of tenderness and pain of a throbbing acute character in the region of the sternum, rigors, sweats, fever and more or less prostration. When the abscess is large enough to encroach upon the air-passages there is cough and dyspnœa, the latter, however, is usually not a prominent symptom. Sometimes there is expectoration which may be bloody. Irritation of the fauces may also be present. The physical signs may be negative, but if the abscess presses beyond the sternum laterally there will be dulness on percussion which gradually increases and later evidences of a tumor. The abscess may break externally or into the trachea, œsophagus, pericardium, pleural cavity or burrow downward into the abdomen. Erosion of the sternum has also been noted.

Chronic abscess of the mediastinum may present the general features of tumor or may be latent. In these cases the symptoms may resemble those of solid tumors. There is often a tendency to spontaneous cure.

The symptoms of abscess of the mediastinum may be masked by those of other conditions, as illustrated by a case of the author's:

The patient, a man of about fifty, was admitted to the Metropolitan Hospital in a condition of mild delirium. No history was obtained, physical examination revealed the presence of pneumonia in the lower lobes of both lungs; there were also symptoms pointing to sepsis. Autopsy showed pneumonia as diagnosed, also a considerable collection of pus in the mediastinum and the presence of a thick yellowish layer or partly inspissated purulent exudate over the pulmonary pleura.

The differential diagnosis includes the consideration of solid tumors and aneurysm. The presence of hectic fever and the more rapid course of the affection point to acute abscess. The presence of a bruit, tracheal tug and diastolic thrill indicates aneurysm. It is very difficult to differentiate between a solid tumor and a chronic abscess. The history of the case and constitutional symptoms will assist, but the aspiration needle is often the sole source of reliance.

The treatment is mainly surgical. Trephining the sternum may be necessary.

TUMORS, SARCOMATA AND CARCINOMATA.

Sarcomata, carcinomata, lymphomata, lympho-sarcomata, hydatids, fibromata, dermoid cysts, lypomata, enchondromata and gummata may all occur, but the forms of neoplasms generally found are sarcomata and carcinomata. Sarcomata are probably the most frequent. The majority of cases are said to occur in the male sex before the fortieth year.

Primary sarcomata may be seated in the thymus gland, lymphatic glands, pleura, lungs or the fibrous tissues of the mediastinum itself. Primary carcinomata may originate in the bronchi, lungs, œsophagus or thymus glands. Secondary malignant growths generally arise in the lymphatic glands.

Symptoms.—Early symptoms of mediastinal tumor are negative. In some instances the tumor remains latent. When symptoms are present they make their appearance gradually and invariably point to pressure as the cause. Substernal pain is more or less constant. It is often accompanied by a sense of oppression and is usually situated in the upper sternal region, but radiates laterally and even down the arms. This is due to pressure upon the bronchial plexus. Dysphagia and dyspnœa are both present in varying degrees of intensity. In some instances there are asthmatic attacks. Some observers mention excessive dyspnœa and rarity of dysphagia. The symptoms obviously vary according as to whether the pressure is upon the trachea, bronchi or œsophagus.

Cough is also a symptom. It may be paroxysmal and metallic in character. There may also be aphonia and hoarseness. If there is implication of the sympathetic nerve or vagus the rate of the pulse may undergo change, being either accelerated or diminished in frequency. Implication of the sympathetic may also cause inequalities of the pupil and local hyperæmia. In some instances there may be spasms of the diaphragm from pressure on the phrenic nerve. If pressure falls upon the subclavian vein or superior vena cava there is cyanosis and œdema of the parts for which these vessels act as venous channels. The inferior vena cava may also be compressed, but less frequently. Other symptoms are expectoration which may be either prune-juice or fetid, hæmoptysis and occasionally there is fever. There may also be implication of the pleura. In addition there will develop sooner or later emaciation, anæmia and cachexia. In the writer's experience pressure symptoms may be very marked long before the development of cachexia.

The variability of the above symptoms obviously depends upon the size of the tumor and its situation. The following classification of Pepper and Stengel as to the latter serves as a useful guide, but in many instances the dividing line cannot be drawn:

1, *When the tumor is situated in the anterior mediastinum, the symptoms being more prominentt than the physical signs:* Here the symptoms point chiefly to pressure on the vena cava, subclavian and right and left innominate veins. There may be thrombosis due to pressure on the yielding walls of the vein, distortion of the veins of the upper extremities, head, neck and upper part of the chest; œdema and lividity and coldness are also present, and the superficial veins become distorted and tortuous. There is pressure on the arteries causing inequality of the pulse at the wrist, on the inferior laryngeal nerve causing hoarseness and aphonia and on the sympathetic resulting in inequality of the pupils. As the tumor increases in size it may press upon the air-passages and cause dyspnœa. The temperature may be normal or subnormal. Cachexia in this variety is usually not so marked.

Physical Signs.—*Inspection* may show the sternum pushed forward and occasionally eroded. *Palpation*, vocal fremitus, negative, increased or diminished. Pulsation is rare, if present it lacks the diastolic shock of aneurysm. Percussion may show an abnormal area of dulness. *Auscultation* may be negative or transmitted respiratory and cardiac sounds may be audible. Occasionally a diastolic bruit may be heard due to pressure on the aorta or pulmonary artery.

II. *When the tumor is situated in the middle and posterior portions of the mediastinum, in proximity to the bronchi, œsophagus, aorta and nerves, and the physical signs are not prominent:* The prevailing symptoms are dyspnœa with very difficult inspiratory effort, pressure on the vena cava with resulting œdema of the abdominal walls and lower extremities, the peculiar paroxysmal cough, metallic, loud, sometimes constant and hacking due to pressure on the pulmonary plexus, with muco-purulent or blood streaked expectoration. Dysphagia is prominent in this group and may be an early and in some cases the only symptom. On the other hand it may be absent. Vomiting, palpitation, irregularity of the heart, syncope from pressure upon the pneumogastric, effusion into the pleura and œdema of the upper part of the abdomen may also be present. The temperature may be moderately elevated, subnormal or there may be high fever. Cachexia is as a rule a marked symptom in this group. The physical signs are of little significance.

III. *When the tumor originates in the pleura or lungs:* In either instances both structures become involved, accompanied by similar symptoms, but the pleura is the most frequent seat of lesion.

The first symptom in this group is pleurisy, and the disease may even be so diagnosed at first. There is sharp pain, cough, difficult respiration and effusion which point decidedly to pleural inflammation. Instead of running the usual course and finally decreasing in severity, the symptoms increase in intensity, especially the pain which extends along the intercostal nerves to the neck and arms. The cough also remains, and the expectoration may become bloody and on examination show traces of the morbid growth. Emaciation is rapid and cachexia pronounced. Secondary growths may appear in the lungs.

Paracentesis is likely to show a bloody or slightly chyliform fluid due to the presence of fatty matter. The latter is found when sarcoma or cancer is present. Another aid to the diagnosis is the unsatisfactory relief to the dyspnœa and the unusual resistance to the trocar. Fränkel speaks of the value in diagnosis of certain swollen cells of an endothelial type which becomes detached and transformed in cases of malignant pleuritic disease.*

Diagnosis.—The striking similarity of the symptoms except in Pepper's third group to those of aneurysm of the arch of the aorta is suggestive. When aneurysm is attended with the characteristic physical signs the diagnosis is without difficulty, but in those cases where pressure symptoms are present and the physical signs negative, the diagnosis in the earlier periods of the lesion is often impossible, and it is only by the subsequent developments that it can be recognized. In the instance of a malignant tumor, emaciation and cachexia sooner or later appear; if there is aneurysm, cachexia is absent, the patient's general condition remains fair and the disease runs a longer course.

X-RAY DIAGNOSIS, see Part VII.

Prognosis.—Death usually ensues in from six to eighteen months.

Treatment.—This is simply palliative. Sustain the strength and administer anodynes. Arsenic may be of service and is about the only remedy for which any claim is made, especially if the growth is sarcomatous.

Other morbid conditions of the mediastinum of more or less rarity are hæmorrhage into the mediastinal tissues which may arise from rupture of an aneurysm or trauma, emphysema from trauma, occurring in whooping-cough and tracheotomy, etc., suppuration and enlargement of the thymus gland. The latter when associated with tumor or abscess of the mediastinal tissues are not distinguishable from the same.

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* System of Medicine, James Tyson, Philadelphia, 1898.

PART V.

DISEASES OF THE DIAPHRAGM.

DISEASES OF THE DIAPHRAGM.

The diaphragm may be affected mechanically by morbid conditions in adjacent structures, or it may be the seat of functional disturbances or organic disease. These conditions may be summarized as follows:

I. Disorders from Mechanical Interference.—Hindrance to the action of the diaphragm may arise in various thoracic and abdominal diseases. The whole structure may suffer or only a part. It may be displaced upwards or downwards. The principal diseases of the chest in which this condition may occur are pleurisy with effusion, pneumothorax, emphysema, tumors, large pericardial effusions and excessive hypertrophy of the heart. The diseases of the abdomen which are also attended with this condition are distention of the stomach, tympanites, peritonitis, pregnancy, large fœcal accumulations and tumors, especially those of the liver, ovaries, spleen and kidneys. The clinical phenomena which result from these obstructive influences are at once manifested by the changes in respiration, which become rapid, labored and superficial. Frequently there is retraction of the lower portion of the chest wall during inspiration, due to the action of the diaphragm. The patient suffers from oppression and inability to take a free breath. He may also complain of dragging pain which is referred to the region of the ensiform cartilage. Coughing is difficult or impossible.

II. Paralysis.—This may occur from mechanical causes, such as the pressure on the origin of the phrenic nerve by tumors, the effect of toxæmia, such as diphtheria and lead poisoning, from hæmorrhages into the cord, injuries and acute ascending paralysis. The extent of the paralysis depends upon the degree of interference which the phrenic nerves sustain. When the upper part of the cord is paralyzed either through injury or disease, there is total paralysis of the diaphragm, when both phrenic nerves are cut or suffer interference from pressure or otherwise, total paralysis also results. If one phrenic nerve is affected, only one-half of the diaphragm is paralyzed.

The symptoms necessarily vary according to the extent of interference. The early symptoms are dyspnœa and a sensation as if breathing was impossible. The respirations are in consequence rapid, superficial and the respiratory movements are confined to the upper part of the chest. An important and diagnostic phenomenon is the reverse movements of the epigastrium and hypochondrium. Instead of bulging during inspiration both regions recede and the abdominal viscera in consequence is not forced downwards.

Functions which call for descent of the diaphragm suffer interference. Sneezing, vomiting, urination and defecation are difficult. Coughing likewise fails to be effectual and in consequence sputum is retained. The lower portion of the lungs becomes hyperæmic, bronchial secretions, not being expelled, accumulate, the air-vessels become filled, atelectasis and pulmonary œdema develop and the patient gradually succumbs.

The rate of development of these symptoms varies according as to whether the onset of the paralysis is rapid or gradual. Paralysis of quick development if total is speedily followed by death. In fact, except in occasional cases arising from diphtheria, death is the usual outcome.

The diagnosis of paralysis of the diaphragm can be made by the character of the respiration and retraction during inspiration as described.

Treatment is frequently without much effect. Artificial respiration and oxygen inhalation should at once be instituted, strychnine hypodermically and alcoholic stimulants should be administered. If the paralysis is unilateral the patient should be instructed to lie upon the healthy side in order to assist the enfeebled and collapsed lung.

III. Spasm.—The diaphragm may be the subject of clonic or tonic spasm. This condition is dependent upon organic lesion of the nerve centres at the origin of the phrenic nerve, direct excitation of the diaphragm or of the phrenic nerve itself, reflex irritation and hysteria.

The most frequent manifestation of clonic spasm is hiccough. This is due to the sudden descent of the diaphragm which causes the air to rush into the open epiglottis. Cough may sometimes be due to clonic spasm. Tonic spasm may arise from strychnine poisoning and rabies.

A form of asthmatic paroxysm is also mentioned by some writers as arising from this condition. In this variety expirations are most difficult and greatly prolonged, while inspirations are short. The lungs are distended, the patient suffers intensely and asphyxiation may seem imminent. Tonic spasm is often attended with severe pain and a sense of constriction in the region of the diaphragm which may be paroxysmal. If the spasm continues and the diaphragm remains fixed, the patient soon dies from asphyxiation.

The treatment varies according to the cause, and is generally unsatisfactory. In the milder forms, especially those of a nervous or hysterical nature, sometimes a sudden reflex irritation will afford immediate relief, as, for example, irritating the nose so as to induce sneezing. Drinking cold water, faradism over the region of the diaphragm and hypochondrium will occasionally relieve. When the cause is gastric irritation, lavage will prove effective.

In hysteria apomorphia gr. ⅛ hypodermically has proved successful by producing emesis. In severe forms morphia sulphate hypodermically and inhalations of chloroform and amyl nitrite are sometimes necessary.

Other remedies are agaricus, cistus, cicuta, coccus, cuprum, hyoscyamus, ignatia, kali brom., laurocerasus, moschus, nux vomica, stramonium and veratrum album.

IV. Inflammation.—The serous covering of the diaphragm, both pleural and peritoneal, may be the seat of acute inflammation. In the case of pleural inflammation there arises the condition known as diaphragmatic pleurisy, which has been discussed under the general subject of pleurisy. In peritonitis the peritoneal covering of the diaphragm may become involved as a part of the general process.

Inflammation of the diaphragm may also occur from traumatism, sepsis and occasionally from causes which cannot be clearly determined.

Chronic inflammation may occur either from extension of inflammation from neighboring organs or from the effect of local irritation. It may ultimately result in fibroid changes in the muscular tissues.

The symptoms of chronic inflammation are usually ill-defined and difficult to determine. There is generally loss of the normal free movements of the diaphragm and the presence of morbid con-

able to cause such change. The indefi
is renders positive diagnosis impossible.
eumatism.—In common with other
m may be the seat of rheumatism. The
by pain referrable to the region of the
aggravated by breathing, especially when
n and relieved by rest. Respirations
All functions calling for action of the
pain.

VI. Morbid Growths and Infections.—Th
be the seat of benign and malignant neoplasms.
In the instance of the latter the disease arises
adjacent structures.

The diaphragm may also be attacked by the tu
trichina spiralis, the echinococcus and the cystic
nition of these conditions is mostly confined to p
ination.

PART VI.

CLUBBING OF THE FINGERS AND PULMONARY OSTEO-ARTHROPATHY.

CLUBBING OF THE FINGERS.

SYNONYM.—*Hippocratic Deformity of the Fingers.*

THIS singular and as yet unexplained phenomenon was known to the ancients, but seems to have been forgotten until attention was directed to it by Trousseau. Clubbing of the fingers is an affection in which the terminal phalanges become bulbous and flattened laterally so as to present a spatula-like appearance, the nails curving over the ends of the fingers towards the palmar surface. The parts involved are the finger pads and tissues about the nail. The bone and periosteum remain entirely free from change. The fingers are livid, but dusky, when there is cyanosis.

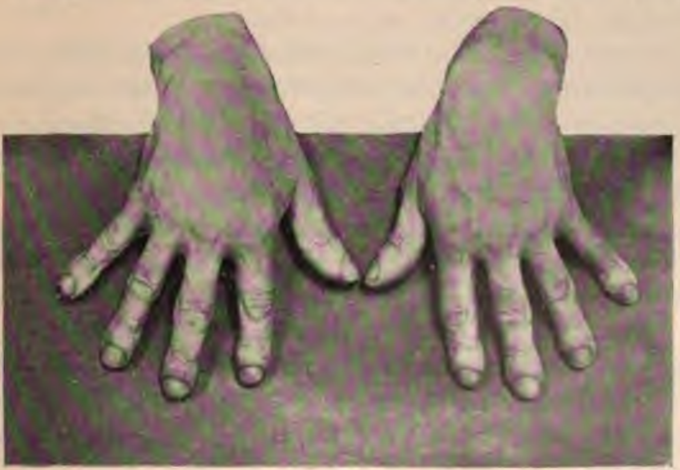

FIG. 56.—Club fingers in a case of pulmonary tuberculosis.

Microscopic examination has not revealed the nature of the changes. The condition usually comes on very gradually without attracting the attention of the patient, but sometimes may develop rapidly. The thumb and index finger appear to be first affected and after these the other fingers. Sometimes the little finger escapes. Sometimes again all the fingers become involved simultaneously. Usually both hands are attacked, although occasionally one alone may show these changes. In rare instances the toes are also involved.

The diseases with which clubbing of the fingers occurs are chronic pulmonary tuberculosis, empyema, asthma, emphysema, bronchiectasis, abscess of the lung and congenital heart disease. It is also found in amyloid disease with suppurative processes without pulmonary lesions and in rare instances in health. Its cause is unknown. According to Buhl it is due to fibrous thickening of the rete-mucosum.

PULMONARY OSTEO-ARTHROPATHY.

This rare affection was recognized by Bamberger in 1889 and Marie in 1890 independently. It was named by the latter *osteo-arthropathie hypertrophante ou pneumique.*

It consists of a multiple osteitis which chiefly affects the terminal phalanges of the hands, feet and extremities of the long bones of the limbs, especially the lower two-thirds and neighboring joints. The result of these changes is enlargement of the parts of the bones involved. The nails are large and curved over the finger ends "like a parrot's beak." The enlargement at the end of the fingers have caused them to be likened to drum sticks. The joints may creak or grate on motion. The elbows and knees may all be similarly affected. The toes may also be affected like the fingers. The rarity of the disease is shown by the fact that Thayer in 1896 collected only fifty-five cases, and in 1892 Newton and Mercelis* reported a case which they stated was only the eighth on record in this country.

The ætiology is undetermined. The thoracic diseases with which it may be associated are chronic tuberculosis, chronic pleurisy and emphysema. It may also occur with syphilis, neoplasms and stomach diseases. Its association with club fingers was observed in seven of Thayer's cases. Messalongo and Thayer† maintain that the pulmonary element is not essential. Thayer found it absent in twelve of his fifty-five cases. From this it may be concluded that the pulmonary factor simply predominates, and that it is absent in nearly one-quarter of all recorded cases.

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* A case of Pulmonary Osteo-Arthropathy, R. C. Newton, M.D., and Elizabeth Mercelis, M.D., International Clinics, 11th Series, vol. iv., 1902.

† Hypertrophic Pulmonary Osteo-Arthropathy and Acromegaly, William S. Thayer, M.D., N. Y. Medical Journal, January 11, 1896.

The pathology is also unknown. The affected bones are enlarged, rarified and show indications of inflammation. The synovial membrane is thickened and the cartilage eroded. The disease has been compared to amyloid degeneration. Marie suggests that toxines from pulmonary disease excite an irritant effect upon the bony and articular structures. Thorburn regards the process as a form of benign tuberculosis. Whatever may be the essential nature of the pathological changes, it is certain that chronic toxic conditions of some kind are always present.

The disease is most insidious in its course. It may appear at any period of life and frequently is accompanied by pain of a rheumatic character.

The diagnosis is usually easy. The only condition with which it might be confused is acromegaly, but osteo-arthropathy does not attack the face which is the case notably with acromegaly. Again in acromegaly the fingers and toes are uniformly enlarged, likewise the wrists and ankle joints, and the soft parts of the joints are not affected.

The course of the disease is protracted. In some instances it may progress slowly to a certain point and there remain without further change.

Treatment is negative. Demons and Beraud have used subcutaneous injection of extract of sheep's lung for which they claimed improvement.*

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* Nervous and Mental Diseases, Archibald Church, M.D., and Frederick Petersen, M.D., 4th Ed., Philadelphia, 1902.

PART VII.

THE ROENTGEN OR X-RAY IN THE DIAGNOSIS AND THERAPEUTICS OF DISEASES OF THE CHEST.

By William H. Dieffenbach, M.D.

Electro-therapeutist to the Flower and Hahnemann Hospitals, New York.

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By William H. Dieffenbach, M.D.

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The discovery of the X-ray by Wilhelm Conrad Roentgen, in 1895, gave to the medical profession an additional means of precision in diagnosis in diseases of the chest, the value of which is now being slowly but surely determined and applied. It will be our purpose to treat of only such methods as have been thoroughly tested and are of proved efficiency.

An X-ray suitable for examinations of the thorax can be secured through the use of a large Ruhmkorff coil of from sixteen- to twenty-spark capacity with the Wehnelt or electrolytic interrupter, or a large modern static machine, which is employed to energize a six-inch or eight-inch Crooke's tube. The relative merits of these two methods have been determined in favor of the coil, more especially for skiagraphic purposes, although a sufficiently powerful static machine in the hands of one thoroughly familiar with its workings can be made to produce good results. The scope of this work precludes delving into the physics of the X-ray, or the construction of the apparatus used for its generation. We will merely mention and explain a few terms which will occur from time to time, so as to make the text more readily understood.

As, up to the present time, the various skiameters have not been generally introduced, we will continue to make use of the old terms, *low*, *medium* and *high*, in speaking of the vacuum of tubes employed and their respective penetrability of substances. A low tube (also called a soft tube) shows the bones of the hand dark in shadow, and expends its action in the superficial tissues. A high tube (also called a hard tube) shows the bones of the hand grayish or white in shadow, and while affecting superficial tissue somewhat, has more penetrating power than the low tube, and is therefore employed preferably for fluoroscopic or skiagraphic purposes. A medium tube is one showing a dark, grayish shadow of the bones of the hand, and its action lies between that of the low and high tubes, depending on the tendency of the vacuum. These terms are somewhat arbitrary, as the vacuum and the tubes undergo frequent change, and the designation of the tube must change accordingly. Testing is required to determine the status of a tube at the time of its employment. In order to obviate dermatitis of the hands of the operator, we have constructed a make-shift skiameter at the Flower Hospital, as depicted in Fig. 57, for use in testing-tubes. This consists of the articulated hand, wrist and forearm of the skeleton attached to a suitable stand, and it admirably suits the purpose for which it is intended ; it can easily be handled and readily determines the relative vacuum of the tubes. Benoist's skiameter,

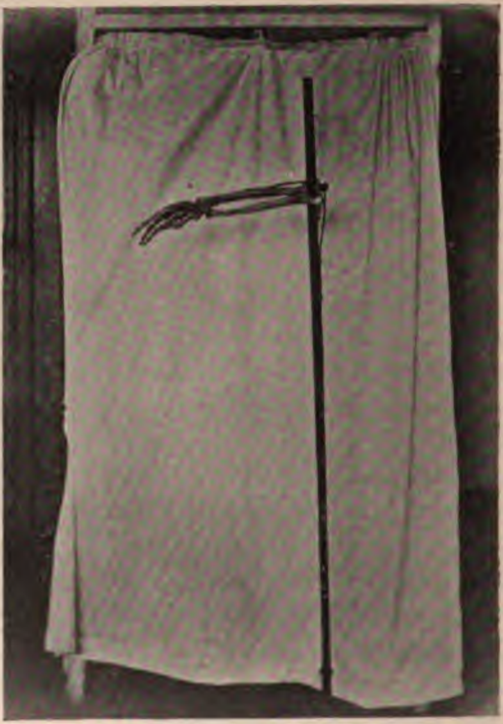

FIG. 57.—Flower Hospital skiameter.

made of various thicknesses of aluminum, and the Walter skiameter are employed by some operators. Dr. J. T. Pitkin, of Buffalo, New York, at the meeting of the American Roentgen Ray Society, at Philadelphia, December, 1903, mentioned a new test for determining the vacuum of tubes. We quote:

"In regard to the relation of the different tubes and the degree of penetration of the rays they are capable of furnishing, valuable information can be obtained by a study of the color markings upon the cavity of the cathode cup. When the circular spot is large, light blue and transparent, with a play of colors at the outer border, the tube is of a high variety. In medium tubes, instead of the light blue centre, it will be of a dark shade of blue, less transparent and smaller in size. In the softer, or lower varieties of tubes, I find either that the central spot is of a golden hue or a very minute, dark, central spot, having a red border. In other words, the size of the circular central spot on the cathode cup increases with use and with the degree of penetration of the rays. Thus, by inspection of the cathode cup of any of my tubes, I can tell what degree of penetration the tube will render. The cathodal spectrum, or play of color, extends from the centre outward with use, the size being proportional to the amount of service that a given tube has seen."

With a good working apparatus and a correctly gauged tube, examinations can be made either with the fluoroscope or screen, or by means of the radiograph or skiagraph. Although, Von Ruck and others have denied the value of the X-ray as an important aid in diagnosis in pulmonary diseases, their information, based on early experiments with inefficient apparatus, has now been proven faulty.

Before undertaking the examination of patients for thoracic diagnosis by means of the X-rays, it is essential to study carefully the normal thorax by means of the fluoroscope or screen and to examine a large number of radiographs or skiagraphs in order to become familiar with normal conditions and deviations from the normal. An excellent skiagram (Fig. 58) will familiarize the reader with the average appearance of the normal thorax. Changes in outline of the heart, aorta, lungs and other structures, indicate some abnormal condition. In fluoroscopic or screen examinations, the patient bares the upper portion of the body and is placed from six inches to twelve inches from the tube; the screen or fluoroscope being applied close to the skin, the centre of the cathode target being directed over the region to be examined. The cathode target should be changed as the fluoroscope or screen ascends or descends, as the best penetration is obtained in line with the cathode target. But few minutes are required for careful examination, the anterior and posterior thorax should be thoroughly mapped out, particular attention being paid to the movements of the diaphragm. The screen or fluoroscope employed is either the barium platinum cyanide or the pure tungstate of calcium. The tube for these examinations should be a tested one of high vacuum.

In examinations of the heart it is well to have the respiratory movements checked at different times by requesting the patient to cease breathing at stated intervals. For securing permanent records of the condition of the thoracic contents the skiagraph is essential. This branch of work requires special skill and attention to detail and is acquired only after some experience. With the present perfected apparatus the chest can be skiagraphed in from one to thirty seconds by means of the coil, and from three to eight minutes with a large static apparatus. The coil, therefore, is preferable, as rapid exposures are necessary in order to prevent interference with the image through constant respiratory action.

To secure a skiagram the apparatus, including the tube, is carefully tested, the patient is placed in a recumbent position with arms extended over the head, a suitable table being employed, a special extra rapid X-ray sensitive plate, size fourteen inches by seventeen inches or eleven inches by fourteen inches, is placed in the plate-holder, below the parts to be skiagraphed. In important cases it is always advisable to make two exposures, or to secure an anterior and a posterior skiagram. The length of the exposure depends on the character of the tube and apparatus and the depth of tissue to be penetrated; the more corpulent the patient the longer the exposure. Plates after exposure should be carefully marked with a case number, date, subject, etc., and developed and fixed *lege artis*, as promptly as possible. If required, prints can subsequently be made from the finished plates, or the latter alone may serve as permanent records of the case. These radiographs can be secured

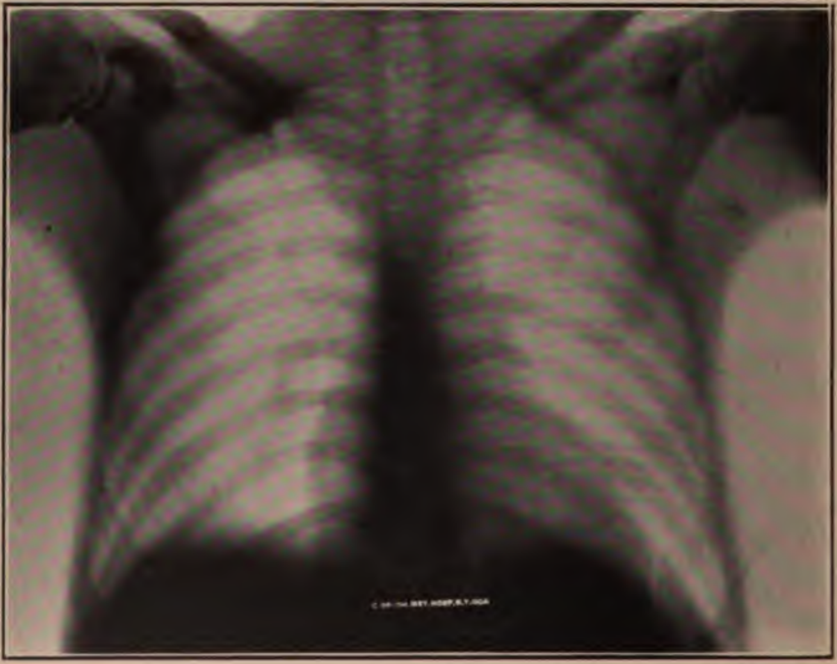

FIG. 58.—Normal chest.

from time to time and the progress of the case noted and permanent records so marked. In tuberculosis, for example, the clearing up of a former hazy zone predicts a favorable prognosis, showing as it does resolution of tissue to normal conditions; on the other hand, increase of the shadows show increased consolidation with modification of the prognosis.

FLUOROSCOPIC (RADIOSCOPIC) AND RADIOGRAPHIC APPEARANCE OF THE THORAX.

If we examine a normal thorax (Fig. 58) we will note a central opacity on both sides of which the lattice work produced by the ribs will be seen, between which are the clear or diaphanous spaces; the central opacity is caused by the vertebral column, the sternum, the large blood-vessels and the heart; the shadows of the latter are noted sometimes to the right, but generally to the left of the median line, and in close contact with the diaphragm; then, by placing the hands of the patient over the head, the scapula is rotated outward and the lung space in the respective regions appears clear. By means of the fluoroscope the heart-beat can be distinctly noted with the movement of the organ against the thoracic walls. The motion of the diaphragm can also be closely studied. This is an important point, the so-called *Williams' Sign* (limited excursion of the diaphragm), which is now recognized as an early sign of involvement of the lungs. Some authors even claim to be able to diagnose a pre-tubercular condition with this sign. The diaphragm can be best studied by placing the fluoroscope or screen at the back of the patient about the sixth or eighth dorsal space, employing a high vacuum-tube. In health, with normal respiration, the diaphragm moves one-half to three-quarters of an inch on either side; in forced respiration the right leaflet of the diaphragm moves two and three-quarters inches, the left leaflet two and a half inches. These figures are an average ascertained through the study of a number of normal chests. The right leaflet in nearly all cases shows fully one-quarter of an inch more mobility than the left.

Williams (*Roentgen Ray in Medicine and Surgery, 1902*) was, we believe, the first to emphasize the fact that in incipient tuberculosis of the lung one of the first signs noted was lack of excursion of the diaphragm of the side affected. This has now been verified so frequently, and is so valuable a diagnostic point, that Williams' Sign should be looked for in all cases of persistent cough, and an early, tentative diagnosis of tubercular invasion will be warranted if this sign is present and the symptoms of other lesions are eliminated.

In consolidation, the fluoroscope will be found much more reliable than the most expert percussion, especially where the lesion is disseminated. In several cases where the apices were affected the author mapped out the affected area by percussion and ausculation, and a fluoroscopic examination invariably showed a larger area of involvement than could be obtained by the usual old physical signs. A central pneumonia can be readily diagnosed by means of fluoroscopic examinations, and questionable cases can be verified by radiography.

Having become familiar with the normal aspect of the heart, blood-vessels and lungs, it will be merely a matter of experience in diagnosing abnormal shadows either with the fluoroscope or screen, or from the radiograph. In treating of the separate diseases, this point will be thoroughly illustrated.

The exact size of the heart can be marked in indelible ink on the chest, or transferred from this sketch to a permanent record, or a record may be kept by means of a skiagram. The progress of cardiac conditions may thus be accurately noted from time to time. Calcareous deposits on the valves have been skiagraphed; arteriosclerosis has been noted in the coronary arteries and atheromatous deposits have been radiographed in the aorta.

DISEASES OF THE HEART.

Dilated or hypertrophied heart can be readily noted, provided we are familiar with the normal appearance of the organ; both conditions present enlargement of the shadow, but the hypertrophy can be distinguished by the rapidity and force of contraction with enlarged left ventricle. The displacement of the heart is downward and to the left.

In pericarditis with effusion the size of the heart is increased, but a differentiation between this condition and simple enlargement must be made from other physical signs. A radioscopic examination, however, shows marked impairment in the cardiac movements, which, combined with the greatly increased area, is fairly conclusive

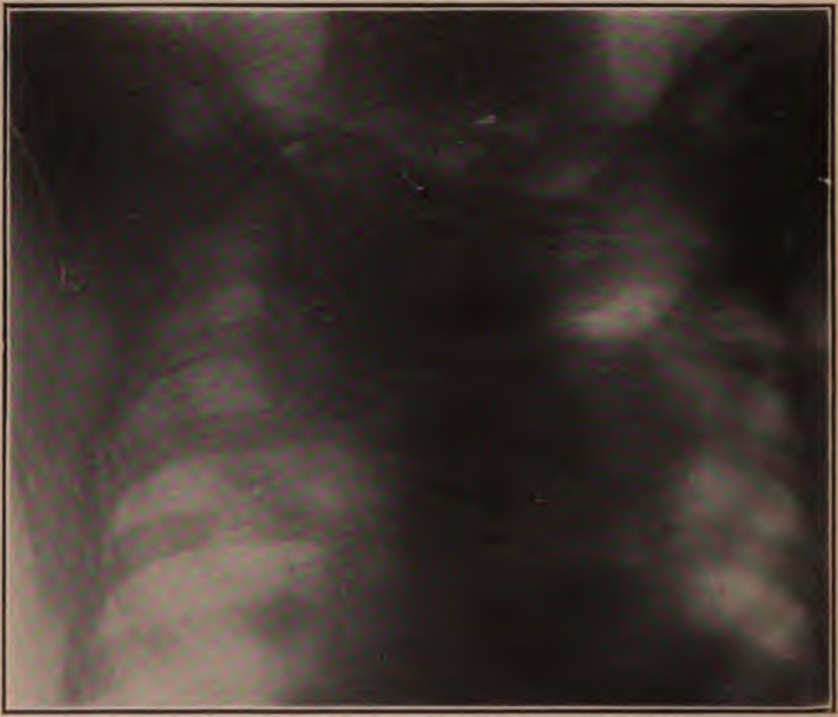

FIG. 59.—Aneurysm of the arch of the aorta, ascending and transverse portion. Figs. 37 and 38 are the sphygmograms of this case.

of pericarditis, if the history of the case corresponds. The general condition of the cardiac muscle can also be noted in many cases, and prognosis can be made with more certainty with the aid of the Roentgen Rays than without it. Therapeutically, the X-ray has not been utilized in diseases of the heart and aorta to our knowledge.

In some cases, calcification of the valves of the heart has been observed.

DISEASES OF THE AORTA.

The outline of the aorta can readily be ascertained either with the fluoroscope or the radiograph. Atheromatous placques have been skiagraphed, but the greatest value of the X-ray has been found in the diagnosis of aneurysm. The accompanying photo-engraving of a skiagram (Fig. 58) shows a case of aneurysm of the aorta with marked dilatation of the heart, and gives the reader a fair idea how these abnormal shadows are pregnant with diagnostic importance.

Aneurysm of the aorta may be confounded with mediastinal or other thoracic tumors, but the well-rounded appearance of the shadow in the former should be looked for, and, if in doubt, a skiagram should be secured.

DISEASES OF THE LUNGS.

Pulmonary Tuberculosis.—In diseases of the lung, more especially in pulmonary tuberculosis, the X-ray has been found of greatest service. Without fear of contradiction, it may be decisively stated that pulmonary tuberculosis can be diagnosed earlier by means of the X-ray than by any agent now at our disposal. Impaired mobility of the diaphragm (Williams' Sign) of the affected side, and the haziness of the affected tissue, instead of the clear diaphanous space, speak eloquently, and are positive indications noted much sooner than when, through breaking down of tissue and purulent decomposition, the sputum appears, and with it the finding of Koch's bacillus. Percussion and auscultation, even the vaunted Krönig method, appear much later than the fluoroscopic or skiagraphic signs; when they do appear the disease is already well established. When the lung is primarily attacked, even before marked changes in temperature are noted, the diaphragm will be found less active on the affected side, and observation from time to time of its increasing immobility or return to normal mobility will assist the observer in rendering judgment as to improvement or aggravation.

In pulmonary tuberculosis the fluoroscope or skiagram shows a grayish or mottled appearance of the parts involved (Figs. 60 and 61). In quite a large number of cases it appears that lesions exist in both lungs, although one side is usually the chief sufferer. This haziness will, if consolidation takes place, become very dark, but if, on the contrary, improvement takes place, a normal, clear or diaphanous space will be noted. Involvement of new areas of infection can be noted, and consolidation, if on the increase, can also be determined and the prognosis made accordingly.

Cavities in the lung can be clearly seen. The fluoroscope shows a clear, usually rounded or but slightly irregular space, surrounded by dark spaces of consolidated tissue, as illustrated in Fig. 62. The area of the cavity appears lighter or almost white. In many cases, if the cavity contains pus or other exudates, the diagnosis is impossible, owing to the fact that the shadows will harmonize more or less with the surrounding consolidation. Suspected cases of pulmonary tuberculosis are examined as follows:

After securing a detailed history, the patient's thorax is bared, and, with the apparatus in flawless condition, the cathodal target of the tube is directed to the apices of the lungs from the dorsal aspect. The tube should be about six inches to eight inches from the patient, and examination should be in a dark room, or a room with black shades, so as to secure a clear shadow. The vacuum of the tube must be medium to high in order to secure penetration. Another advantage of using higher vacuum-tubes is that dermatitis is less frequent than with low tubes. The screen or fluoroscope is placed close to the skin, and the hand manipulating the same should be properly protected, either with tin-foil lined gloves or by means of a shield attached to the fluoroscope or screen. Bearing in mind the transparency of the normal lung, deviations of the same can be noted, and if a record is desired it can be sketched on prints of the normal thorax, secured for this purpose, or simply noted on the case record. These deviations of transparency may be of several degrees: First degree, slight haziness; second degree, increased haziness; third degree, consolidation or obliteration of transparency. In miliary tuberculosis the lung presents a charac-

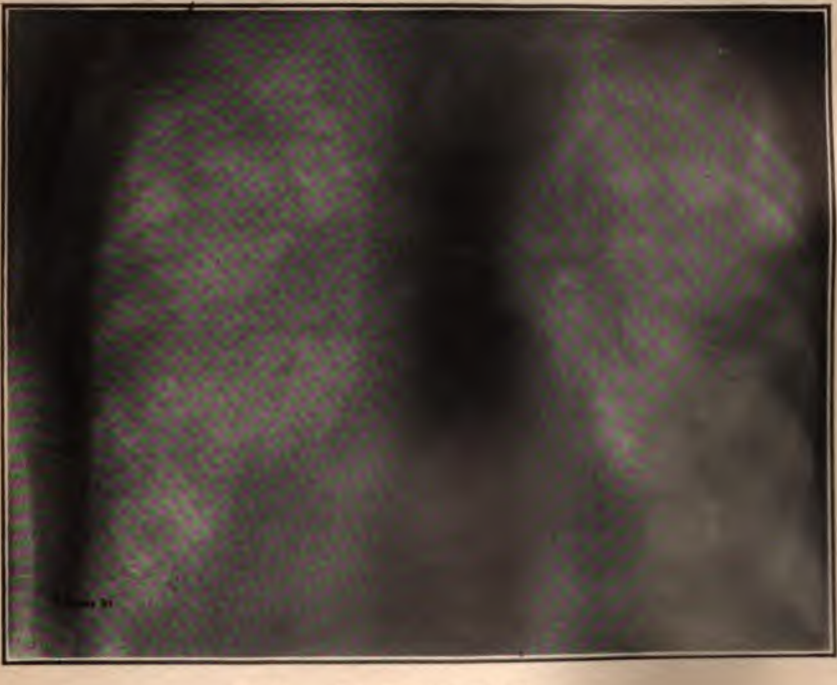

FIG. 60.—Pulmonary tuberculosis.

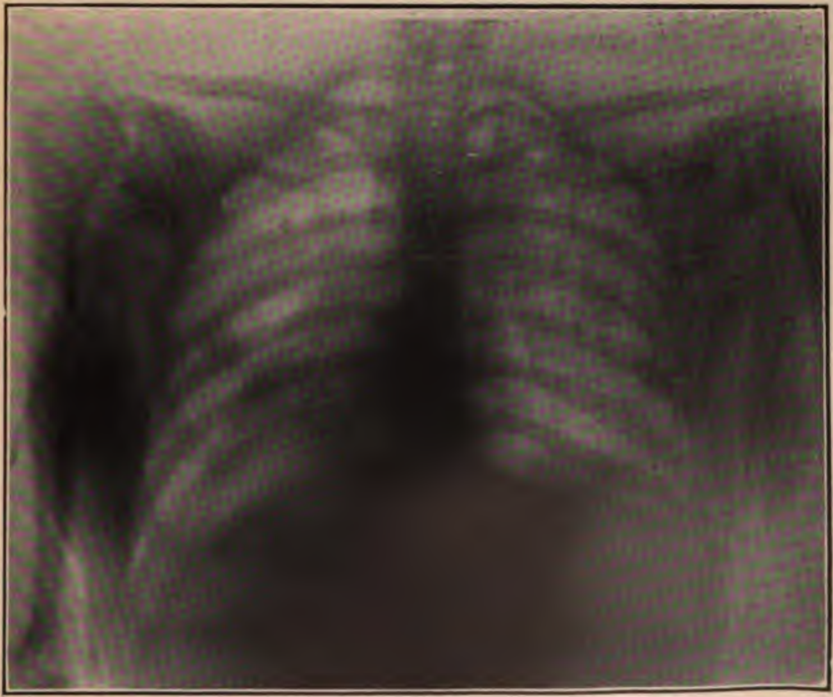

FIG. 61.—Pulmonary tuberculosis, showing cavities in upper lobe, right of illustration.

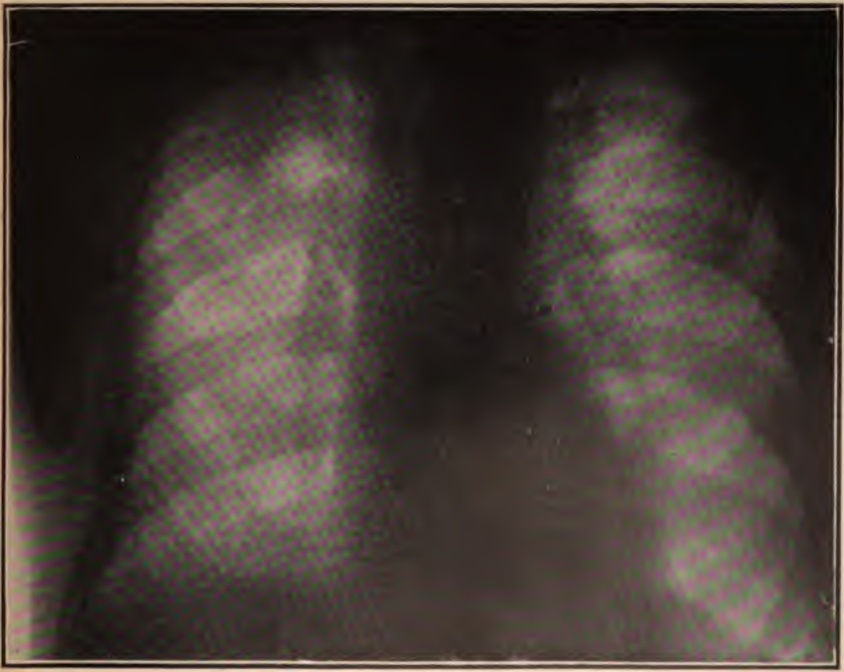

FIG. 62.—Pulmonary tuberculosis.

teristic mottled appearance, which, when once seen, can be readily recognized again. Records of fluoroscopic surveys are particularly valuable for purposes of prognosis, as the progress of the disease can be accurately noted.

The upper portion of the lung having been examined, the tube is now directed over the central portion of the thorax and both sides carefully viewed and conclusions noted. The operator must recall the normal appearance of the great vessels and heart, and incidentally observe the size, position, character of contraction and movements of the heart; the transparency or lack of transparency of the lower lobes should also be noted. Cavities, if such exist, when not filled with pus or exudates, can be determined by the peculiar white color of the parts affected, surrounded by an area of opacity.

Having examined the lobes of the lung the diaphragm is now examined and deviations in excursion, that is, Williams' sign, noted; bearing in mind that the right side normally has an excursion of one-quarter of an inch more than the left. The side most affected would have decided immobility. This diagnostic point has been observed in a large number of cases before other evidences of pulmonary lesions were positive. Careful practice in the method of examination is therefore incumbent upon all who desire to acquire proficiency in diagnosis, especially in the early diagnosis of pulmonary tuberculosis.

The value in prognosis of this method has already been touched upon. In many cases it is desirable not only to examine anteriorly, but also posteriorly, the thoracic changes being noted in each case.

We have personally employed the X-ray for the past four years in all tuberculous cases which we have treated and can state that the improvement noted since the employment of the X-ray in these cases has been more marked than before, all other methods remaining practically the same. We have sought for a reason for the good effects noticed after the use of the X-ray and have come to the following conclusions:

The X-ray, like all other forms of light, if sent through the tissues for a limited time, provokes distinct stimulation of the parts through which it passes; if the action is kept up too long, this stimulation is succeeded by dermatitis and subsequent necrosis. The bility of giving, therefore, short exposures only cannot be too strongly emphasized. The stimulation of the X-ray to the tissues induces hyperæmia with accompanying leucocytosis, and, if there is anything in the Mentchnikoff theory, phagocytosis takes place with destruction of bacterial activity. Another theory advanced is, that the X-ray sets free ozone along its course; the ozone in turn being inimical to bacterial existence.

Another, not unimportant feature, is the psychic effect on the patient. The patient knows and feels that something is being done for him besides the ordinary hygienic and medicinal measures, and the encouragement he thus receives has a decided beneficial effect on the buoyancy of his spirits.

We employed the X-ray originally in a case of combined phthisis and intestinal tuberculosis, in which all hope had been abandoned. No literature on the subject being at hand, we worked on the following technic, based on the principles of therapeutics outlined above: The patient bared chest and abdomen, and was exposed anteriorly for five minutes in the thoracic and abdominal regions respectively; and then exposed posteriorly over the thoracic region for five minutes, a high tube being employed, placed from four inches to six inches from the body. One area at a time was usually treated, the tube being placed in line with a new area at each treatment, until the whole chest had been thoroughly covered. In cases where the lesion is confined to but one section, of course, this section alone will receive the most attention. We gave treatments every other day, and the patient was directed during the treatments to take deep breathing exercises, so as not to inhale the ozone set free from the static machine during the operation.

Pulmonary Emphysema.—In emphysema the screen or fluoroscope shows the characteristic barreled shaped thorax, the ribs being in a horizontal position during forced inspiration. The heart is usually in a vertical position; this being due to the lowering of the diaphragm and increased pressure on the lungs. The diaphragmatic excursion in emphysema is limited, especially in the upper area. The normal rise of the dome of the diaphragm being absent. Portions of the lung present a whitish, absolutely transparent appearance of the parts affected, surrounded by an area of less transparency or even a slight opacity.

Asthma.—Asthma shows no distinct radioscopic or radiographic features. Frequently, abnormalities of the osseous structure have been noted, and during an attack of asthma the excursion of the diaphragm is inhibited. It resumes its function after the attack has subsided.

Pneumothorax, Hydro- and Pyo-Pneumothorax.—The fluoroscope in these cases shows the diaphragm on the affected side to be practically immobile, the lung tissue showing in the former a whitish transparency; in the latter condition the black, dark outline of the fluid will be noted with a superincumbent white layer of the air.

Hæmoptysis.—Reasoning from the beneficial effects obtained in hæmorrhage of uterine cancer, Dr. H. P. Deady has used the X-ray in pulmonary hæmorrhage, in the "so-called pulmonary bleeders." He states that he makes it a practice to submit all acute hæmorrhagic cases to this treatment as early as the conditions permit. (E. G. R.)

Croupous Pneumonia.—As already mentioned, we have in the X-ray a method for accurate diagnosis of central pneumonia, a condition usually otherwise arrived at only by exclusion. The central area affected would show a dark shadow of distinct consolidation. In the early stages of pneumonia, before consolidation has taken place, the fluoroscope shows commencing haziness of the parts affected; and when consolidation supervenes a distinct and irregular shadow will be seen. Comparison of the two sides reveals marked difference in opacity. The diaphragm is limited on the side affected unless the lesion is bilateral, when total immobility of the diaphragm presents itself.

The progress of the case in pneumonia can be noted with precision and resolution watched; when the diaphragmatic mobility increases improvement can be predicted, and with resumption of mobility to normal condition complete restoration will be noted.

Differentiation between pleurisy with effusion and pneumonia by means of the fluoroscope is made generally on the character of the opacity of the tissue affected.

In pneumonia, as already stated, the opacity is irregular in outline, and the healthy lung tissue, owing to increased work thrown upon it, looks particularly white. In pleurisy with effusion the outline of the opaque region is well defined, and the opposite lung shows normal transparency; it also shows restriction in breathing instead of hyperactivity of the well side, as noted in pneumonia.

Gangrene of the Lungs.—In suspected cases of gangrene of the lungs the region affected would show a well marked opacity with infiltration into the surrounding tissue. If surgical measures are to be invoked, the exact area to be operated upon can be accurately mapped out upon the thoracic wall before operative procedures take place.

DISEASES OF THE PLEURA.

In cases of pleurisy without effusion, no change in the transparency of the thoracic shadows is noted, although diaphragmatic immobility of the affected side is noted at once. When effusion has taken place opacity will be seen, the rise or fall of the liquid being rapidly determined by the fluoroscope or screen. The position of the heart can also be determined, a left-sided pleurisy with effusion tending to displace the heart markedly to the left and upward. The fluid, whether serous, sero-fibrous or purulent, usually presents a regular, sharp outline and is homogenous throughout. Some radiographers claim to be able to differentiate the shadows of a sero-fibrous and a purulent effusion; exact differentiation, however, is somewhat difficult.

TUMORS OF THE THORAX.

Carcinomata, sarcomata, echinoccocus cysts, actinomyces, mediastinal tumors, glandular hypertrophy and other enlargements can be noted, although diagnosis in some cases is difficult. These tumors must be differentiated from cardiac hypertrophy and aneurysm of the aorta; in the latter, the location and the rounded appearance of the tumor, combined with other physical signs, will be fairly conclusive.

TRANSPOSITION OF ORGANS.

Transposition of the organs in the chest can be seen by means of the fluoroscope, if a permanent record is desired a skiagram may be taken. A case of transposition of the heart to the right side was recently seen by the author, the mediastinum appearing particularly large.

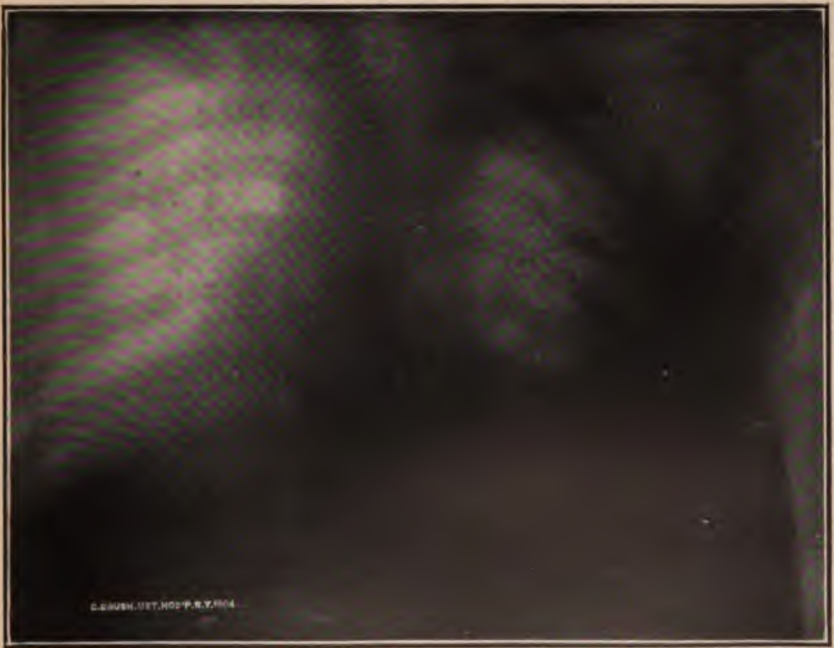

Fig. 63.—Pleurisy with effusion.

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